THE UNIVERSITY OF TEXAS MEDICAL BRANCH NUMBER
Galveston, Texas

Foreword

The University of Texas Medical Branch was established in Galveston in 1890 by legislative decree "as an institution of the first class." The nucleus of its original faculty consisted of four well trained young men between 28 and 30 years of age who established the Departments of Anatomy, Surgery, Therapeutics, and Pathology. The first Professor of Surgery, Dr. James E. Thompson, became well known for his work on cleft lip and palate surgery and for his treatise on the surgical approach to the long bones. He was succeeded in 1927 by Dr. A. O. Singleton, who initiated the residency system here six years later. Under his guidance and leadership the Department has grown to include five divisions for Board Certification training with a four-year period in General and Thoracic Surgery and three-year periods in Orthopedics, Genito-Urinary Surgery, Neurosurgery, and Plastic and Maxillofacial Surgery. The Department as a whole, under the present Chairmanship of Dr. Robert M. Moore, is anxious to stimulate resident interest in teaching and research and provides an opportunity for presentation of papers on clinical and experimental subjects at a weekly staff conference and in cooperation with the Post-Graduate Division at formal and informal courses given under their auspices.

The members of the Department of Surgery wish to thank the editors of *The American Surgeon* for the privilege of sponsoring the August issue of the journal. The articles with few exceptions are the work of instructors, residents, and fellows and deal with both clinical and investigative phases of their work. It is our wish that this University of Texas Number be dedicated to the memory of Dr. A. O. Singleton, under whom most of us have served as associates and students and whose words and influence are ever present with us.

T. G. Blocker, Jr., M.D.
Professor of Surgery
(Plastic and Maxillofacial)

The University of Texas Medical Branch
Galveston, Texas

THE
AMERICAN
SURGEON

Subscription in the United States, $8.00

Vol. XVIII, No. 8 August, 1952

EXTRASKELETAL OSTEOCHONDROSARCOMA

Review of the Literature and Report of a Case

LEE W. SHAFFER, JR., M.D.
Galveston

EXTRASKELETAL ossifying malignancies are infrequent. Wilson,[17] in 1941, reviewed the literature and found only 30 cases and added an additional 7. Since that time 7 cases have been reported in the American literature.[1,4,6,13,15,16,18] Table 1 gives a summary of these cases.

Mayer and Friedman[11] reported a case of a soft tissue, bone-forming tumor in the gastrocnemius muscle which was thought to be an osteogenic sarcoma. However, the benign clinical course led to a reexamination of the pathologic tissue with the final diagnosis of a benign extraskeletal bone-forming tumor. Three similar cases were reported by Wilson.[17] One of these was a calcified neurofibroma and two were fibrous osteomas. Three cases of sarcoma that developed in areas of myositis ossificans are not included in this review since myositis ossificans is usually attached to bone and should not be considered a true extraskeletal lesion.[12]

Malignant ossifying tumors, no doubt, are more frequent than indicated from this review. Cases will go unobserved unless the title indicates the presence of bone.

It is of interest to observe in the 44 cases of extraskeletal osteogenic sarcoma previously reported and the 1 case included in this paper that 32 of the cases occurred in females and 13 in males. The anatomical sites of the neoplasms were: 12 cases in the breast, 8 in the thyroid, and 7 in the thigh. There were 2 cases in each of the

From the Department of Surgery, Orthopedic Division, University of Texas School of Medicine, Galveston.

following locations: kidney, leg, upper arm, and gluteal region; and one in the chest wall, abdominal wall, mesentery, meninges, lip, urinary bladder, brachial plexus, and gallbladder. The site was unknown in 1 case.

The age distribution would suggest that these tumors are more common in young individuals and in the older age group.

The age at which 43 tumors were observed was as follows:

10-19 years — 2 cases
20-29 years — 7 cases
30-39 years — 5 cases
40-49 years — 2 cases
50-59 years — 13 cases
60-69 years — 9 cases
70-79 years — 5 cases
(Age was not given in 2 cases)

The clinical course in these neoplasms varies widely. Some were present for only a few weeks or months, while others were present as long as 20 years before removal. Local recurrence was observed in 8 of 45 cases, and 12 cases terminated with widespread metastases. It is of interest to note that in 2 cases there were no metastases at the end of two years, and six months respectively.[14] Wilson,[17] in citing these two cases, stated that no metastases were present at 10 and 7 years. Parsons and Henthorne,[13] in reviewing Wilson's paper, have suggested that the tumors in these 2 cases may have been benign. Turner and Craig's[16] case of meningeal osteogenic sarcoma showed no evidence of recurrence after two years. It is well known that benign meningeal tumors may show bone formation.[5]

The pathologic features of these extraskeletal malignant tumors, like the clinical manifestations, vary widely. In many of these cases osteoid tissue was not suspected. The four characteristic histologic components of these tumors are spindle cells, cartilage, osteoid tissue, and true bony trabeculae. However, not all of these elements may be present in any given tumor. These characteristics are nicely illustrated in the paper by Wilson.[17]

The problem of histogenesis in these cases has provoked much speculation. The embryonic rest theory as well as the theory of blood stream transference of osteoblasts apparently has been discarded.[19] Mallory[10] mentions the possibility of periosteal origin, but points out that this could not explain cases of ossification in laparotomy wounds. This is also borne out by the not infrequent occurrence of ossification in other tissues widely separated from

TABLE 1

Author	*Patient and Complaint*	*Character of Lesions*	*Remarks*
Parsons and Henthorne[13]	A 53 year old man. Two centimeter ulcer on right side of lower lip. Right submental nodes enlarged.	Pleomorphic spindle cells, giant tumor cells, osteoid tissue, and bony spicules.	Right radical neck dissection and removal of lesion. Removal of recurrence three years following first operation. No metastases. Recurrence six months after second operation with fixation to mandible. Third operation—resection of right mandible and portion of floor of mouth. Expired 24 hours postoperatively. DIAGNOSIS: Osteogenic sarcoma.
Tremblay, Crane, and Harris[15]	A 69 year old man. Hematuria and dysuria with large grayish-yellow mass in bladder.	Spindle-shaped tumor cells, osteoid material, osteoclastic giant cells, and cartilage cells.	Cystotomy and local removal. Died four and one-half months later of urinary infection and uremia. Autopsy revealed osteogenic sarcoma of bladder with metastases to peritoneum, lungs, and intestinal serosa. Believed to arise in remnants of Wolffian body in region of the trigone. DIAGNOSIS: Osteogenic sarcoma.
Hamer and Wishard[6]	A 76 year old man. Hematuria and urinary frequency. Large tumor in right upper abdomen. Gastric ulcer.	Tumor in right kidney 15 cm. in diameter containing atypical bone spicules and irregularly arranged osteoblasts.	Treated with irradiation and multiple transfusions. Expired one month later of hemorrhage from gastric ulcer. Autopsy showed pulmonary metastases from renal tumor. DIAGNOSIS: Sclerosing osteogenic sarcoma.

TABLE 1 (*continued*)

Author	*Patient and Complaint*	*Character of Lesions*	*Remarks*
Woltman, Adson, and Abbott[18]	A 29 year old woman. Pain, weakness, and wasting of left arm following trauma to left shoulder two years previously. Nine years later complete brachial plexus paralysis and Brown Sequard syndrome.	Fibroblastic and osteoblastic cells lying in a fibro-osteoid matrix. Mature bone.	Cervical laminectomy 11 years following trauma. Osteogenic sarcoma compressing cord at level of C-5 and surrounding brachial plexus. DIAGNOSIS: Osteogenic sarcoma grade I.
Turner and Craig[16]	A 37 year old woman. Weakness right hand, convulsive seizures, and inability to speak.	Tumor in postfrontal and parietal region contained new bone, osseous and fibroblastic tissue with frequent mitoses.	Progressive improvement of neurologic signs after removal. No evidence of recurrence two years later. DIAGNOSIS: Meningeal osteogenic sarcoma.
Batts[1]	A 71 year old woman. Swelling in left neck. Nervousness, hyperhidrosis, and weight loss. BMR, plus 31.	Calcification in thyroid tumor containing osteoid tissue and bone.	Patient died four months later of cardiac failure. DIAGNOSIS: Osteoid-osteo fibrosarcoma.
Couret[4]	A 55 year old woman. Mass in left breast, not fixed. No palpable nodes.	Contained spindle cells, osteoid tissue, and abnormal mitoses.	Patient symptom-free one year later. DIAGNOSIS: Osteoid sarcoma.

periosteum such as the heart valves and aorta. Tremblay, Crane, and Harris[15] believe that in their case the tumor arose from a Wolffian body remnant in the region of the bladder trigone. Most authors[3,9,10,14,17,19] emphasize the totipotency of fibroblastic tissue and its ability to differentiate into any mesodermal tissue when given the proper environmental and chemical stimulus. Leriche and Policard[8] even postulate that all osteogenic sarcomas are connective tissue sarcomas in which there has been osseous metaplasia. Binkley and Stewart[2] are inclined to believe that many of these cases appear to be extraskeletal osteogenic sarcomas only because of cellular infiltration into areas of ischemic hyalinization. However, they cannot explain the presence of cartilage.

Fig. 1a. Tumor involving left biceps muscle.

Case Report

C. J. M., a 26 year old Negro man laborer, was admitted to John Sealy Hospital complaining of a progressive enlargement of his left upper arm. This swelling was first noticed one year prior to admission. The patient attributed it to trauma which was sustained when he was thrown from a moving auto-

mobile six months prior to the onset of the swelling. Pain first occurred in the left arm two months before hospitalization and persisted and radiated to the left shoulder and lower neck.

Fig. 1b. Preoperative roentgenogram of the left humerus showing irregular calcification in the tumor. Diaphysis intact.

On examination a firm, smooth, 10 by 10 cm. mass was present over the upper portion of the left biceps muscle (fig. 1a). The tumor apparently was not attached either to the humerus or to the overlying skin. It was slightly warmer than the surrounding tissue and nonpainful to palpation. A roentgenogram of the left humerus showed a soft-tissue density with centrally located irregular areas of calcification (fig. 1b). The diaphysis was intact. Roentgenograms of the chest were normal.

The tumor proved to be a sarcoma by biopsy section and a left interscapulothoracic amputation was performed on the fourth day of hospitalization. Postoperative bone survey was normal, and he was discharged 16 days following the operation.

The pathologic specimen consisted of the entire left arm and shoulder girdle. In the biceps muscle there was a mass 8 by 8 by 5 cm. which extended to the tendinous portion of the long head (fig. 1c). On cut surface the tumor was firm, grayish-white and glistening. Local areas of degeneration were filled

with a clear mucoid material. Although the tumor was located adjacent to bone, it was not continuous with the periosteum. There was no gross extension of the tumor into the shoulder joint.

Fig. 1c. Sagittal section of specimen showing tumor in biceps muscle not attached to humerus.

Histologic sections from various areas of the tumor showed a variety of mesothelial tissues (fig. 2). Some were composed predominately of irregular cartilage cells in a mottled spongy matrix. There was an area of well formed osteoid tissue and an area showing mature bony trabeculae. More cellular areas of the tumor were composed of irregular hyperchromatic spindle cells which showed numerous mitoses. Blood vessel invasion was demonstrated in several sections. No metastases were present in the axillary lymph nodes.

Fig. 2a. Section showing tumor giant cell and spindle cells. Hematoxylin and eosin. 413X.

Fig. 2b. Section showing skeletal muscle invasion by tumor cells. Hematoxylin and eosin. 160X.

The patient returned for examination six weeks following amputation. The operative wound was well healed, and he complained of only minor discomfort in the scar. A roentgenogram of the chest was interpreted as normal. The patient did not return to the clinic for approximately six months, at which time he had a moderate amount of edema of the face and extremities. A productive cough, left pleuritic pain, dyspnea, and orthopnea had been present for three weeks. Roentgenograms of the chest showed an area of increased density,

10 by 6 cm., in the base of the left lung, and a similar lesion, 1 cm. in diameter, in the base of the right lung. A mass, 3 cm. in diameter, was present in the anterior superior mediastinum. Roentgen irradiation was considered to be contraindicated even for palliation at this time. At latest report, eight and one-half months following surgery, the patient is somewhat improved subjectively, although confined to bed, and no doubt death will follow from the metastatic lesions.

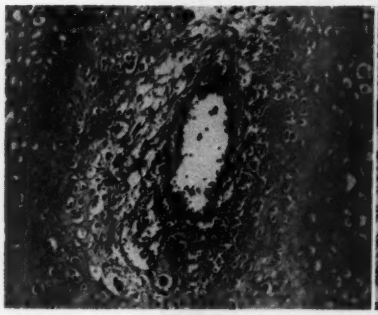


Fig. 2c. Section showing irregular cartilage cells. Hematoxylin and eosin. 160X.

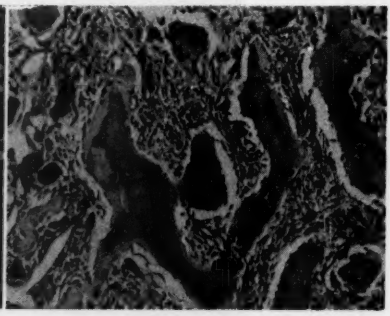


Fig. 2d. Section showing bony trabeculae of membranous type. Hematoxylin and eosin. 150X.

SUMMARY

1. A case of osteochondrosarcoma arising extraskeletally in the biceps muscle of a 26 year old Negro man is reported.

2. Seven cases of extraskeletal ossifying malignancies reported in the American literature since 1940 are summarized.

3. Age and sex distribution, anatomic location, and clinical course are correlated in the 45 known cases.

4. The theories of extraskeletal ossification are reviewed.

REFERENCES

1. Batts, M.: Osteogenic sarcoma of thyroid gland, Am. J. Surg. *49:*390 (Aug.) 1940.
2. Binkley, J. S., and Stewart, F. W.: Morphogenesis of extraskeletal osteogenic sarcoma and pseudo-osteosarcoma, Arch. Path. *29:*42 (Jan.) 1940.
3. Broders, A. C., and Pemberton, J. deJ.: Primary osteogenic sarcoma of thyroid gland, Surg., Gynec. & Obst. *58:*100 (Jan.) 1934.
4. Couret, J. S.: Osteoid sarcoma of breast, New Orleans M. & S. J. *98:*412 (March) 1946.
5. Cushing, H. W., and Eisenhardt, L.: Meningiomas, Springfield, Ill., Charles C Thomas, 1938, p. 50.
6. Hamer, H. G., and Wishard, W. N., Jr.: Osteogenic sarcoma involving right kidney, J. Urol. *60:*10 (July) 1948.
7. Kretschmer, H. L.: Retroperitoneal pararenal osteoma, Surg., Gynec. & Obst. *67:*108 (July) 1938.
8. Leriche, R., and Policard, A.: The Normal and Pathological Physiology of Bone, St. Louis, C. V. Mosby, 1928.

9. Mallory, F. B.: The Principles of Pathologic Histology, Philadelphia, W. B. Saunders, 1914, pp. 276, 650.
10. Mallory, T. B.: Group of metaplastic and neoplastic bone and cartilage containing tumors of soft parts, Am. J. Path. *9:765*, 1933.
11. Mayer, L., and Friedman, M.: Extraskeletal bone-forming tumor of fascia, resembling osteogenic sarcoma, Bull. Hosp. Joint Dis. *2:*187 (Oct.) 1941.
12. Pack, G. T., and Braund, R. R.: Development of sarcoma in myositis ossificans, J.A.M.A. *119:*776 (July 4) 1942.
13. Parsons, W. H., and Henthorne, J. C.: Extraskeletal osteogenic sarcoma; report of case of osteogenic sarcoma of lip, Ann. Surg. *119:*595 (April) 1944.
14. Rhoads, C. P., and Blumgart, H.: Two osteoblastomas not connected with bone, histologically identical with osteogenic sarcoma and clinically benign, Am. J. Path. *4:*363 (July) 1928.
15. Tremblay, R. G.; Crane, A. R., and Harris, A.: Primary osteogenic sarcoma of bladder, J. Urol. *51:*143 (Feb.) 1944.
16. Turner, O. A., and Craig, W. M.: Osteogenic sarcoma of meningeal origin, Arch. Path. *32:*103 (July) 1941.
17. Wilson, H.: Extraskeletal ossifying tumors, Ann. Surg. *113:*95 (Jan.) 1941.
18. Woltman, H. W.; Adson, A. W., and Abbott, K. H.: Neuritis ossificans with osteogenic sarcoma in brachia plexus following trauma; report of case, Journal Lancet *66:*372 (Nov.) 1946.
19. Woolley, P. G.: Heteroplastic bone and bone marrow formation, J. Lab. & Clin. Med. *1:*502 (April) 1916.

MEGACOLON—FROZEN SECTION CONTROL OF SURGICAL TREATMENT

O. T. Kirksey, Jr., M.D.
John H. Childers, M.D.
Galveston

Since Swenson's work[4,5,6,7] on megacolon, there has been interest in resection of the rectum and lower sigmoid colon with an end to end anal pull-through anastomosis for radical cure. Lee[3] has reviewed the literature and gives an excellent bibliography.

Whitehouse and Kernohan[8] reviewed the literature on the myenteric plexus in megacolon and studied 11 cases of their own. They concluded that absence of the ganglion cells in the rectum and lower sigmoid colon was a constant significant morphologic change. Bodian, Stephens and Ward[1] confirm this work on the basis of 28 cases.

At the John Sealy Hospital, we have had 4 cases of megacolon in which a Swensen resection was done with good results. In the last 3 of these cases, the presence or absence of ganglion cells was determined by frozen section technic during the operation. In our cases, we were able to do this and the subsequent paraffin sections confirmed our frozen section findings. This microscopic control of the extent of the resection is valuable because Whitehouse and Kernohan[8] have shown that 20 per cent of the time the ganglion cells are absent well up into the dilated hypertrophied sigmoid colon. Bodian, Stephens and Ward[1] found that ganglion cells were often absent as high as 5 cm. into the dilated colon. If a colostomy has been done, often the sudden dilatation of the colon will disappear and cannot be used as a landmark. Swenson[7] reported one failure in which the rectum and sigmoid were removed. Later roentgenologic examination showed that the lesion extended to the splenic flexure. A second resection gave good results. The histopathology of this specimen was not given.

Since atypical cases occasionally occur, and in most cases the cells are absent in a variable length of the distal dilated colon, at the suggestion of Dr. Edgar Poth, the laboratory of Dr. Israel Diamond[2] was consulted regarding the use of frozen section control. We have used this method which is essentially as follows:

After the bowel is freed, it is usual to remove a part of the proximal segment of the gut before pulling the sigmoid through the anus. This specimen is then examined by frozen section technic. A

From the Departments of Pathology and Surgery, University of Texas School of Medicine, Galveston, Texas.

longitudinal block 2.0 cm. long is taken from the upper end of the colon. A section 10 to 15 microns thick is cut and stained immediately with toluidine blue. If no ganglion cells are seen, two more blocks are cut at this level and studied.

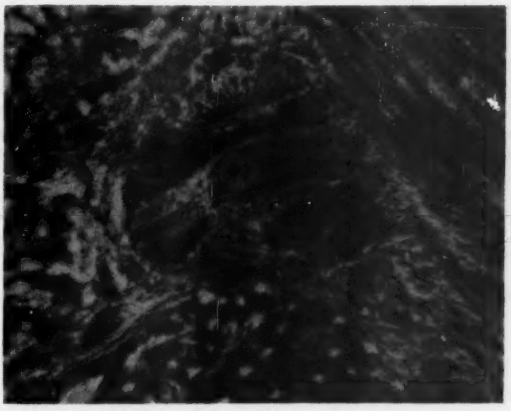


Fig. 1. Frozen section 10-15 microns thick by 630, toluidine blue stain. The ganglion cells are larger than in a paraffin section and the Nissl substance can be seen.

We have had 3 cases studied as indicated above. If the first block showed ganglion cells, all the others at that level also showed these cells. If the ganglion cells of Auerbach's plexus were absent, then subsequent blocks at that level did not show these cells. Thus the surgeon can be told if his resection will be adequate. No special stains or technic are necessary other than those available in a laboratory equipped to do frozen sections.

SUMMARY

1. Our material confirms the constant morbid absence of ganglion cells in the rectum and to a variable extent up into the dilated sigmoid colon in megacolon.

2. Frozen section control of the adequacy of the colon resection is simple, rapid, and reliable and should improve the end results of surgical treatment.

REFERENCES

1. Bodian, M.; Stephens, F. D., and Ward, B. C. H.: Hirschsprung's disease, Lancet *1*:19 (Jan. 7) 1950.
2. Latta, H.: Personal communication to the authors.
3. Lee, C. M., Jr., and Bebb, K. C.: Pathogenesis and clinical management of

megacolon with emphasis on fallacy of term "idiopathic," Surgery *30:*1026 (Dec.) 1951.

4. Swenson, O., and Bill, A. H., Jr.: Resection of rectum and rectosigmoid with preservation of sphincter for benign spastic lesions, producing megacolon, Surgery *24:*212 (Aug.) 1948.
5. Swenson, O.; Neuhauser, E. B. D., and Pickett, L. K.: New concepts of etiology, diagnosis and treatment of congenital megacolon (Hirschsprung's disease), Pediatrics *4:*201 (Aug.) 1949.
6. Swenson, O.; Rheinlander, H. D., and Diamond, I.: Hirschsprung's disease; new concept of etiology. Operative results in thirty-four patients. New England J. Med. *241:*551 (Oct. 13) 1949.
7. Swenson, O.: New surgical treatment for Hirschsprung's disease, Surgery *28:*371 (Aug.) 1950.
8. Whitehouse, F. R., and Kernohan, J. W.: Myenteric plexus in congenital megacolon; study of eleven cases, Arch. Int. Med. *82:*75 (July) 1948.

PRIMARY HYPERPARATHYROIDISM

With Three Case Reports

M. N. Davis, M.D.
Galveston

PRIMARY hyperparathyroidism due to adenomatous or hyperplastic changes of the parathyroid glands is being reported more and more frequently, so that it is not and should not be a rare and unfamiliar clinical entity.

Albright[1] divided hyperparathyroidism clinically into four categories:

1. With bone disease but without kidney disease.
2. With bone disease and kidney disease.
3. Without bone disease but with kidney disease.
4. Without either bone or kidney disease.

In 1948 at Mayo Clinic, Black[3] reported 63 cases of hyperparathyroidism. Twenty per cent showed generalized osteitis fibrosa cystica, 20 per cent had both renal complication and osteitis fibrosa cystica, 20 per cent had renal disease with mild osteoporosis, and 35 per cent had urinary tract disease alone. He concluded that approximately 40 per cent of the cases show evidence of osteitis fibrosa cystica, and about 75 per cent show renal disease with calcification. Hyperparathyroidism existing without renal or osseous changes is rare (about 1 per cent) and is usually referred to as the gastrointestinal type.

Suspicion of hyperparathyroidism should be aroused in every case of nephrolithiasis or nephrocalcinosis. Certain bone changes and symptoms related to hypercalcæmia require an adequate examination of the calcium and phosphorus levels in the blood and urine. It is helpful to know the action of parathyroid hormone in order to understand the clinical manifestations of hyperparathyroidism. Albright believes that the parathyroid hormone first affects the phosphate so that it is dissolved in the body fluids in such a way as to be more easily excreted in the urine. The calcium phosphate equilibrium becomes undersaturated; to remedy the loss of phosphate resorption of the calcium phosphate salts from the bone takes place. With the higher calcium serum level there is increased calcium excretion in the urine. Unless there is an adequate gastrointestinal absorption of calcium, the undersaturated body fluids will again call on bone resorption to correct the equilibrium. In view of the per-

From the Department of Surgery, Urological Division, University of Texas Medical Branch, Galveston.

centage occurrence of the four groups, calcium balance is probably maintained more frequently from the dietary intake of calcium.

The demonstration of the characteristic blood and urine findings is many times equivocal or misleading. Hypercalcemia, hypophosphatemia, hypercalcinuria, and hyperphosphaturia are the criteria for diagnosis of hyperparathyroidism. Blood levels for calcium above 10.5 mg. per 100 cc. and inorganic phosphorus below 3 mg. per 100 cc. should be regarded as exciting suspicion.

It has been demonstrated by McLean and Hastings[6] that relative hypercalcemic states can be present in blood which has subnormal levels of total serum proteins.

In many cases a Sulkovitch test is sufficient to determine the presence of elevated calcium in urine. In equivocal instances a quantitative determination of the urinary calcium excretion must be done while the patient is on a low calcium diet. A low calcium diet containing approximately 125 to 130 mg. of calcium per day should be started three days prior to urine studies. Usually patients with hyperparathyroidism will excrete over 200 mg. of calcium per day, but findings of 150 to 200 mg. are considered significant by many investigators. The normal 24 hour excretion of phosphorus in the urine is reported to be approximately 1 Gm. Repeated laboratory studies are necessary in any suspicious case.

It is significant that the spinal fluid calcium level in hyperparathyroidism is usually within the normal limits of 4.5 to 5.5 mg. per 100 cc., while in other diseases causing hypercalcemia there will be an elevated spinal fluid calcium level.[5]

Hypercalcemia, *per se*, produces nausea and vomiting, muscular weakness and fatigability. Polydipsia and polyuria are probably due to renal damage, but they have been reported in cases without renal calcification.

The bony changes associated with hyperparathyroidism are characteristic if a suspicion of the disease is in mind. In generalized osteitis fibrosa cystica there is a decrease in the bone tissues as a whole because of the continued bone resorption. On roentgenograms there is generalized decalcification of the skeleton with coarse bony trabeculation. The flat bones of the skull show the characteristic "ground glass" appearance. Additional local changes may occur in any part of the bony skeleton. They consist of bone cysts, tumors, and fibrocystic changes. Deformity of the long bones is more common than fracture. With osteoblastic activity the serum alkaline phosphatase will be increased. The lamina dura surrounding the teeth is characteristically absent.

The renal complications associated with hyperparathyroidism are most frequent and important. Albright[1] believes that 5 per cent of all renal calculi or of cases of calcinosis are the result of hyperparathyroidism. In a recent report by Beard and Goodyear,[2] of 150 cases of renal stone disease studied, 8 per cent had hyperparathyroidism proved by operation. Certainly bilateral and recurrent renal calculi and cases of nephrocalcinosis deserve study for the possibility of parathyroid hyperfunction.

In differentiating osteitis fibrosa cystica from other metabolic bone diseases, it is necessary to determine the serum level and urinary excretion of calcium and phosphorus. Osteoporosis, osteomalacia, Paget's disease, solitary bone cyst, multiple myeloma, metastatic malignancy, and Boeck's sarcoid are generalized or localized bone disorders which may be confused with hyperparathyroidism.

The only adequate treatment for hyperparathyroidism is surgical removal of the hyperfunctioning gland or glands. Several authors have claimed good results from roentgenotherapy, but Albright[1] believes that this form of treatment "should be used as a last resort, if used at all."

The surgeon must be able to locate the glands, differentiate their appearance, and understand the physiology and pathology in the more serious cases. Black[3] in his 1948 report of 63 cases found 5 per cent had multiple adenoma and 5 per cent had hyperplasia. Many cases have been reported where the parathyroid adenomas have been located in the mediastinum.

The surgical problems related to identification and removal of parathyroid adenomas have been presented admirably in the publications of Churchill and Cope.[4]

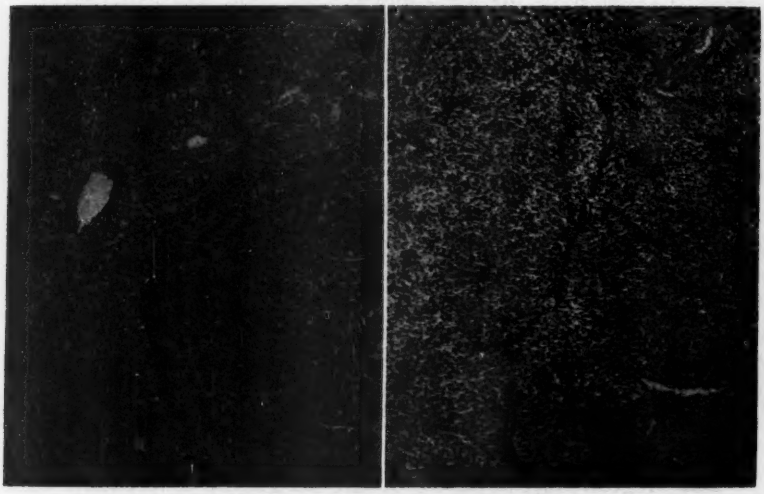
Carcinomatous changes occurring in the parathyroid gland are rare. The symptoms are the same as in adenomatous or hyperplastic growths.

Case Reports

Case 1. H. W. E., a 36 year old white man, was admitted in November 1948 on the Genito-Urinary Service because of bilateral renal calculi. His past history revealed that in 1942 a calculus was removed from the right ureter, and in 1947 more stones were taken from the right kidney. The physical examination was essentially normal.

Urinalysis disclosed a low specific gravity, pyuria, and microscopic hematuria; from urine cultures *Staphylococcus albus* and *Alcaligenes fecalis* were isolated. The blood analysis showed nonprotein nitrogen to be 31 mg. per 100 cc., total serum proteins 6.1 Gm. per 100 cc , serum calcium 11.8 mg. per 100 cc., serum phosphorus 3.85 mg. per 100 cc., and alkaline phosphatase 2.55

Bodansky units. The hemogram was normal. Repeated serum calcium levels varied from 10.8 to 11.8 mg. per 100 cc., while the average phosphorus level was 2.3 mg. per 100 cc. after omitting the admission finding of 3.85 mg. per 100 cc. The urinary output of calcium for 24 hours on a 125 mg. calcium-containing diet was 350 mg.



Case 1, fig. 1. Photomicrograph (150X) through one pole of the parathyroid gland showing preponderance of clear cells in a pseudo-glandular arrangement. By Dr. H. W. Neidhardt (formerly with Department of Pathology).

Case 2, fig. 2. Photomicrograph (150X) showing the large cells with clear cytoplasm. There is tendency for an alveolar type of arrangement, but no glands are seen. Other sections showed small cells with moderate nuclear pleomorphism and hyperchromatism with occasional mitotic figures. There was a tendency for the cells to invade the thick stroma capsule. The question of malignancy could not be satisfactorily settled by the microscopic appearance. (Dr. H. W. N.)

Roentgenologic examination revealed a large staghorn calculus in the left kidney and three small calculi in the right kidney with bilateral pelvic hydronephrosis. Also present were multiple small prostatic calculi. Chest and long bone survey did not reveal any abnormality. Roentgenograms of the skull were not made.

In December 1948 a left pyelolithotomy was performed, and the patient was discharged in January 1949. The patient was instructed to return in two months but did not return until May 1949. The calcium and phosphorus blood values were essentially the same as on the previous entry. In May 1949 a parathyroid exploration was done. A 15 by 8 mm. enlarged parathyroid gland was found near the inferior right thyroid pole. Microscopic study of the tissue removed showed preponderance of oxyphil and clear cells. In some areas, especially at one pole of the gland, clear cells were preponderant. The pathologic diagnosis was moderate hyperplasia of the parathyroid gland (fig. 1).

In June 1949 a right pyelolithotomy was done. This patient has had good

urinary function since that time, and to date there has been no recurrence of renal calcification.

CASE 2. V. B., a 36 year old white housewife, was admitted on the Oto-laryngology Service in February 1949. The patient stated that for several months she had a productive cough which was worse upon rising in the morning. The sputum was usually clear but recently had become blood-tinged; this she attributed to excess blowing of the nose and coughing, sinusitis having been present for several years. Previous roentgenograms of the sinuses showed a "skull tumor" which was diagnosed by a neurosurgeon as a meningioma. The patient also complained of much fatigability and stated that she was very tired upon arising.

Physical examination was essentially normal except for moderate clubbing of the fingers. The past history was of interest in that she had a left renal calculus removed eight years previously in another hospital.

Bronchoscopic examination revealed a marked compression of the trachea beginning just below the level of the larynx. Fluoroscopic examination of the esophagus revealed compression posteriorly and to the right. Roentgenogram of the chest revealed normal lung fields, but in the lateral left thoracic cage there was irregularity in one of the ribs with several punched-out areas. Roentgenograms of the skull revealed a large area of calcification in the right fronto-parietal region. There were also small punched-out areas in the right parietal bone, and diffuse miliary decalcification. The lamina dura was noted to be present. Cervical and dorsal spine roentgenograms showed considerable proliferative arthritic disease.

The only laboratory studies obtained were two urinalyses with normal results except that the specific gravity was 1.006 and 1.004. Hemogram was normal.

Diagnosis was tumor of the thoracic inlet. Exploration was done in March 1949, and a dense mass of tissue was located behind the lower pole of the right thyroid lobe. The mass was fairly well encapsulated. Pathologic diagnosis was a clear cell adenoma of the parathyroid gland. (See comment in figure 2.)

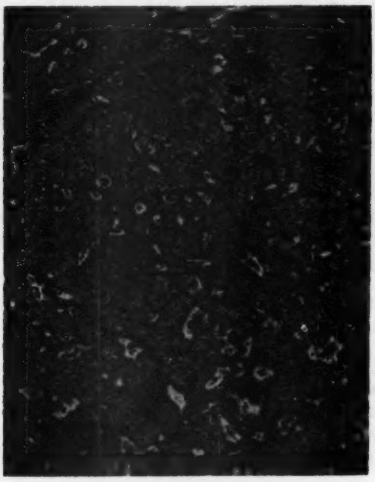
The postoperative course was very stormy, characterized by "mental confusion and postoperative psychosis." Four days postoperatively the serum calcium was 9.75 mg. per 100 cc. She was discharged on the sixth postoperative day only to develop convulsions in her sleep that night. Calcium gluconate was injected intravenously with some relief, but she soon developed another tonic convulsion. Intravenous sodium luminal and paraldehyde finally gave her relief after several convulsive periods. The serum calcium was 7.85 mg. per 100 cc. after the calcium gluconate injection. She was discharged two weeks later without any mental disturbance.

Follow-up visits reveal that the patient now is doing well.

CASE 3. W. L. McC., a 49 year old white housewife, was admitted on the Genito-Urinary Service in July 1951. For the previous four or five years the patient had experienced attacks of chills and fever followed by dull aching pain in the left lower abdominal quadrant radiating to the left costovertebral region. Bladder symptoms were also present. She had a roentgenogram made by her physician and was told that she had bilateral kidney stones. Physical examination was noncontributory.

The urinalysis revealed albuminuria, microscopic hematuria, and gross pyuria. Coliform organisms were isolated in the urine culture. The hemogram showed moderate secondary anemia.

Blood chemical determinations disclosed the nonprotein nitrogen to be 21 mg. per 100 cc., total serum proteins 7.0 Gm. per 100 cc., serum calcium 8.3 mg. per 100 cc., serum phosphorus 2.4 mg. per 100 cc., and the alkaline phosphatase 2.6 Bodansky units. Subsequent repeated calcium determinations disclosed consistent levels between 11 and 12 mg. per 100 cc. The serum phosphorus levels averaged 2.5 mg. per 100 cc., spinal fluid calcium was 4.6 mg. per 100 cc., calcium excretion levels per 24 hours on a 125 mg. calcium daily diet were 184 and 178 mg., and the 24 hour phosphorus excretion levels were 605 and 230 mg.



Case 3, fig. 3. Photomicrograph (150X) showing the clear cells situated in sheets and cords; however, several acini are present. By Dr. John H. Childers (Department of Pathology).

Roentgenograms showed multiple renal calculi bilaterally. A calculus was also present in the gallbladder. Roentgenograms of the chest, skull, and hands did not show any abnormality.

A right pylelolithotomy was done in August 1951. The presumptive diagnosis of hyperparathyroidism was made, but further blood examinations were requested before surgical exploration. She was discharged from the hospital and returned in December with left renal colic accompanied with fever and chills. A left pyelolithotomy was done. Repeated blood and urine determinations were done with essentially the same results as on previous examinations. Because of her weakened general condition, a parathyroid exploration was not advocated; and she was again discharged. She returned to the Surgery Service in February 1952. Neck exploration revealed a 26 by 11 by 5 mm. mass located below the right lower thyroid lobe. Microscopic examination revealed

an encapsulated lesion composed of clear cells situated in large sheets and cords; however, small acini were present. The pathologic diagnosis was parathyroid adenoma (fig. 3). The patient was discharged on March 1, 1952.

SUMMARY

The criteria for the diagnosis of primary hyperparathyroidism are presented. Repeated laboratory determinations are essential in equivocal cases. All patients with renal calcification should be studied for the possibility of hyperparathyroidism.

Three cases of primary hyperparathyroidism are reported. Two patients had bilateral renal calculi not associated with bone changes. The third patient had bony lesions without renal calcification although a renal calculus had been removed eight years before.

REFERENCES

1. Albright, F.; Aub, J. C., and Bauer, W.: Hyperparathyroidism; common polymorphic condition as illustrated by seventeen proved cases from one clinic, J.A.M.A. *102:*1276 (April 21) 1934.
2. Beard. D. E., and Goodyear, W. E.: Hyperparathyroidism and urolithiasis, J. Urol. *65:*638 (Nov.) 1950.
3. Black, B. M.: Surgical aspects of hyperparathyroidism, Surg., Gynec. & Obst. *87:*172 (Aug.) 1948.
4. Churchill, E. D., and Cope, O.: Parathyroid tumors associated with hyperparathyroidism; eleven cases treated by operation, Surg., Gynec. & Obst. *58:*255 (Feb. no. 2A) 1934.
5. Howard, J. E.; Carey, R. C.; Rubin, P. S., and Levine, M. D.: Diagnostic problems in patients with hypercalcemia, Tr. A. Am. Physicians *62:*264, 1949.
6. McLean, F. C., and Hastings, A. B.: Clinical estimation and significance of calcium-ion concentration on blood, Am. J. M. Sci. *189:*601 (May) 1935.

PORPHYRIA AND ITS SIMULATION OF SURGICAL CONDITIONS

E. H. Rentschler, M.D.
Galveston

The symptomatology of some types of porphyria may resemble that of surgical diseases of the abdomen and possibly of the nervous system. The purpose of this paper is to present a summary of porphyria as derived from a review of the recent literature and to present two brief case summaries illustrating the problem of differentiation of this condition from surgical disease.

Porphyria may be defined as a familial, constitutional disturbance due to an inborn error of pigment metabolism in which abnormal quantities of porphyrin compounds are excreted in the urine and feces. The word "porphyria" is derived from the Greek "porphura," meaning "purple," which color may be imparted to the urine of an individual excreting increased amounts of porphyrins in the urine.

Recognition for first establishing the clinical entity of porphyria is given generally to Gunther, who originally described the condition in 1911, although description of some of the clinical features had been made as early as 1871 by Hoppe-Selye.[72]

Hans Fischer in 1924 was the first to report synthesis of the porphyrin compounds and determination of their structural relationships. The basic structure is made up of four pyrrole rings linked together by four methene bridges containing eight replaceable hydrogen atoms. Propionic, acetic and methyl groups are substituted for the hydrogen atoms and by rearrangement of the position of these substitution groups four possible isomers (etioporphyrins) are obtained—referred to as Types I, II, III and IV porphyrins. Types II and IV have never been found to occur naturally, but Types I and III are similar to such compounds as the respiratory enzymes (catalases, peroxidases, and cytochromes), hemoglobin, myohemoglobin and chlorophyll.

The classification of the porphyrias proposed by Waldenstrom in 1937, which has been accepted by most authors, divides the disease into the following types:

1. Congenital;
2. Acute (Idiopathic and Toxic) and
3. Chronic.

From the Department of Surgery, Division of Neurosurgery, University of Texas Medical Branch, Galveston.

The acute type is the one most often coming to surgeons for consideration for operation. The congenital and chronic types will also be presented briefly for purpose of completeness.

Congenital Porphyria

The congenital type is more common in males with onset in infancy or early childhood. The principal features are the excretion of red urine (containing Type I porphrin predominately), pink discoloration of teeth later changing to red-brown, marked photosensitivity of the skin with epidermolysis bullosa or hydroa vacciniforme type lesions resulting in considerable scarring, hirsuitism, hypertrichosis, enlargement of the liver and spleen, and anemia. Treatment for this type is to avoid sunlight, to wear protective clothing and to utilize ointments to protect from the sunlight's ultraviolet rays. Death in these patients is usually due to intercurrent diseases. The congenital type would not come to the attention of surgeons ordinarily, although there has been a report of benefit being given to a patient with this type of porphyria by splenectomy.[4]

Acute Porphyria

The acute type is subdivided into the idiopathic and toxic types, the latter due to the use of some drug by the patient. The symptomatology in both types is the same with the exception of the identification of an etiological agent in the latter type.

The acute type occurs mainly in females with a ratio of 4:1 (females to males). Onset is usually between the third and fifth decades of life and in the idiopathic variety a familial relationship has been observed. It is thought that the trait is transmitted as a Mendelian dominant characteristic.

Abdominal pain is the symptom which frequently causes the patient to seek medical attention. It may be generalized or localized to any part of the abdomen. It may or may not radiate. Nausea and vomiting are experienced, and, typically, severe constipation or obstipation is noted. In rare instances diarrhea is experienced. On examination of the abdomen, muscle spasm, tenderness on palpation and rebound tenderness are usually absent. This is an important differential point in distinguishing porphyria from an acute surgical abdomen. Findings compatible with ileus may be observed. Roentgenologic examination of the abdomen may reveal dilatation of the stomach, small and large bowel; however, there may be hypermotility instead of ileus, with contraction of the stomach and intestine.

It is important to ask about the color of the urine in taking a

history from the patient. Typically the urine is red, portwine-colored or Burgundy wine-colored. This is usually due to the presence of Type III porphyrins, although porphyrins of Type I may be present. However, not all of those patients with acute porphyria will have abnormally colored urine. In many cases there may be excreted the colorless chromogen-porphobilinogen, which will turn pink, red or red-purple in color on exposure to the sunlight. A screening test known as the Watson-Schwartz test (outlined in detail below) may be utilized as a diagnostic aid.

The findings of hypertension and tachycardia are consistently present during an acute attack. There may be moderate elevation of the temperature. Oliguria may be noted and occasionally mild jaundice is observed.

The clinical picture may not end with the symptoms and signs as noted above. If there is involvement of the central nervous system a galaxy of neurologic abnormalities may be noted, including: delirum, evidences of depression, mania and hallucination; involvement of cranial nerves III, IV, VI, VII, IX, X, and XII, with ocular and facial palsies, amaurosis, dysphonia and dysphagia; Jacksonian or generalized seizures; stupor or coma; ataxia and nystagmus; and peripheral nerve and spinal cord involvement, including peripheral neuritis with foot drop and wrist drop, paresthesias with back and leg pain but with no demonstrable loss of sensation on examination, and, typically, an ascending type of flaccid paralysis (with absent tendon reflexes). Any and all of these may be present in a patient with nervous system involvement. Such involvement carries a grave prognosis, especially if bulbar involvement is present. The mortality with this type becomes 90 per cent whereas if the symptomatology is limited to the abdomen alone the mortality is said to be 20 per cent.

Laboratory findings include a moderate leukocytosis by peripheral blood study with the presence of porphyrins in the urine. Electrolyte evaluation reveals a marked lowering of the serum sodium and chloride in some of these patients, but with a normal or low CO_2 combining power. It is not felt that this is secondary to the vomiting inasmuch as no alkalosis exists with this hypochloremia. Adrenal cortical function studies have been done on these patients and no abnormality has been noted. The present opinion is that the hypochloremia and hyponatremia are the result of renal changes in which there is an excessive loss of chloride and sodium in the urine. The spinal fluid is characteristically normal although several cases have been reported where the protein content was elevated. Electrocardiographic changes may be noted but these are not diagnostic.

As has been stated previously, the acute toxic type has the same symptomatology as the acute idiopathic type, except that there is a history of use of some drug precipitating the attack. A list of drugs which have been incriminated at one time or another includes acetanilid, barbiturates, sulfonamides, sulfonal, trional, arsenicals, alcohol, phosphorus, selenium, and lead. If the use of any of these is suspected, the appropriate diagnostic tests may be utilized.

Diagnosis

The diagnosis of acute porphyria is made by identification of increased amounts of porphyrins in the urine utilizing the following methods:

1. Exposure of the urine to ultraviolet light (sunlight) which will cause it to turn to a pink, red or red-purple color, indicating the presence of the colorless porphobilinogen.

Note: Urine should be made acid in order to do this clinical test; alkaline urine may not be affected by the sunlight even if porphyrin compounds are present.

2. Watson-Schwartz test for the presence of the colorless porphobilinogen, performed in the following manner:

(a) 1 cc. of Ehrlich's reagent is added to 1 cc. of urine.

(b) 2 cc. of saturated solution of sodium acetate is added to this and the mixture is shaken.

(c) If pink color develops, add 2 cc. of choloroform and shake vigorously. If the color is due to porphobilinogen, it will not be extracted from the watery phase.

3. Spectroscopy (by the biochemist). The porphyrin compounds exhibit characteristic specific absorption spectra in acid solution.

4. Identification of the type of porphyrin present (by the biochemist), by determination of the melting point of the crystalline form of the methyl esters of the porphyrin compounds obtained from the urine of the patient.

Differential Diagnosis of Acute Porphyria

Increased porphyrin excretion may be noted in other disease conditions as follows: hemolytic jaundice, pernicious anemia (following a reticulocyte response to liver therapy), acute hepatitis, alcoholic cirrhosis, miliary tuberculosis, fungus and bacterial infections, hemochromatosis, and pellagra. It is evident, then, that the excretion of porphyrins, *per se,* without the clinical symptoms and findings, requires further investigation as to the etiology.

Other conditions causing the excretion of red urine must be considered. The urine of anyone taking pyridium or eating red beets may be red-colored. The alkaline urine excreted by those taking or eating phenolphthalein, rhubarb, senna, cascara or santonin may be colored red. The possibilities of hemoglobinuria and hematuria should also be considered.

The differential diagnosis of the various abdominal conditions simulated by porphyria includes: appendicitis, peritonitis, pancreatitis, perforated peptic ulcer, cholecystitis, biliary colic, bowel obstruction, renal colic, pelvic organ disease and pheochromocytoma.

It is not infrequent to find that these patients have undergone previous abdominal exploration with no abnormality observed other than a marked spasm of the bowel musculature. In 1 case reported[66] the appendix, gallbladder, right ovary and right kidney (all found to be normal except for a cyst of the ovary) were removed at separate operations before the diagnosis of porphyria was finally made.

To complete the differential diagnosis various neurologic disorders simulated are hysteria, psychoses, neuroses, Guillain-Barré syndrome, poliomyelitis, familial periodic paralysis, serum paralysis, periarteritis nodosa, encephalitis, progressive muscular trophy and lead poisoning.

Treatment of Acute Porphyria

The treatment of the acute porphyric is largely supportive. If a toxic agent has been found, it should be withdrawn immediately. Other than this adequate fluid, diet and vitamin intake should be maintained. Intravenous calcium may be helpful to alleviate temporarily the symptoms referable to the abdomen. Liver extract is said to help in the acute episode but this is questionable. Pain relief is most consistently obtained from demerol. Prostigmine has been given for relief of the ileus with questionable effectiveness, and one author[52] has reported good relief from abdominal symptoms with the use of intravenous procaine, which effectiveness may be attributed to the vasodilatation, analgesic and anticontracting actions of the drug.

Prognosis of Acute Porphyria

Prognosis for survival for an attack of acute porphyria is 50 per cent for all cases with 90 per cent mortality if neurologic involvement is present and 20 per cent with abdominal involvement alone.

Pathology of Acute Porphria

The pathologic changes noted involve the liver and kidneys and the central nervous system. These are not specific and are for the most part degenerative changes. In the nervous system degenerative changes involve the peripheral nerves, dorsal root ganglia, sympathetic ganglia, anterior horn cells and posterior horn cells of the spinal cord, vagal and hypoglossal nuclei, cells of the white matter and cerebral cortex, Purkinje cells of the cerebellum and the dentate nucleus.

Involvement of the kidneys and central nervous system is attributed to spasm of blood vessels with ischemia to the region. To support this is the observation microscopically of marked spasm and secondary aneurysmal dilatation of capillaries of the fingers and spasm of retinal vessels observed during acute episodes. It is thought that the hypertension present is the response from spasm of blood vessels generally, perhaps to circulating porphyrin compounds. Experimental evidence supporting this is the fact that injection of hematoporphyrin compounds intravenously in animals results in a gradual increase of the blood pressure above normal followed by a fall in blood pressure and death.

The abdominal symptoms are thought to be due to involvement of the sympathetic ganglia and the vagal nuclei with the latter probably responsible for the greater share of the response of the gastrointestinal tract. Another factor may be a direct response of the bowel musculature to circulating porphyrins. As noted above, spasm of the bowel has been noted in these patients at the time of surgery. It has been shown experimentally in animals that application of porphyrin compounds directly on bowel musculature results in marked spasm.

Chronic Porphyria

The chronic type of porphyria is often considered to be an intermediate type in that it has some of the features of the congenital and the acute types. It is more common in females and an association with diabetes and liver disease (particularly cirrhosis occurring with alcoholism) has been observed. The symptomatology includes mild photosensitivity with hyperpigmentation and roughening of the skin of exposed areas, excretion of red-colored urine (containing Types I and III porphyrins) and occasionally jaundice. There may be abdominal symptoms as well as neurologic symptoms as seen in the acute type but these usually are not prominent. Treatment for these is to avoid exposure to sunlight and to treat the liver disease and diabetes. Prognosis is variable with a possibility

of death of the patient with any exacerbation, particularly if neurologic involvement occurs.

Case Summaries

Case 1. A 22 year old white man was admitted to John Sealy Hospital on the General Surgery Service on Sept. 20, 1951, with the complaint of abdominal pain, nausea and vomiting and constipation of three days' duration, with localization of pain to the right lower quadrant of the abdomen on the day of admission. On examination it was thought that there was rebound tenderness referred to the right lower abdominal quadrant. White blood cell count was 18,780 per cu. mm. with neutrophiles, 82 per cent and Stab forms 9 per cent. His temperature was normal. Roentgenogram of the abdomen revealed dilated loops of small bowel suggesting the existence of intestinal obstruction. The patient was operated upon on the day of admission. An appendectomy was performed. No abnormality of the appendix was observed grossly or microscopically.

On the second postoperative day the patient had a generalized convulsion following which he was stuporous for several days. Weakness of his arms and legs was noted. He complained of leg pains also. The patient was then transferred to the Neuro-Psychiatric service. It was then that urine studies were done and the diagnosis of acute idiopathic porphyria was made. During the period following the development of neurologic symptoms and signs the blood pressure rose from 120/80 to 150-180/100. A tachycardia of 120 beats per minute was observed at this time as compared to a pulse rate of 72 noted on admission.

The patient survived this acute episode and eventually left the hospital at his own request, although he had marked difficulty in the use of his extremities due to weakness at the time he went home.

Case 2. A 39 year old white woman was admitted to John Sealy Hospital on Jan. 15, 1952 on the Neurosurgical Service as a private patient. She had been referred by her home physician for study of the possibility of a brain tumor. She had experienced episodes of cramping abdominal pain beginning about Dec. 15, 1951. An intestinal obstruction or pelvic disorder was suspected and laparotomy was performed by the referring doctor on December 20. No evidence of intestinal obstruction was found. A hysterectomy was performed at this time. Following this the patient continued to complain of abdominal pain and severe constipation. On Jan. 4, 1952 she experienced a generalized convulsion. She had one or two similar seizures during the next week. In addition to the abdominal pain she complained of back and leg pain. She exhibited evidence of anxiety, delirium and depression at various times. Drug therapy included Dilantin, Gantrisin and barbiturates.

On admission at John Sealy Hospital neurologic examination revealed stupor, lethargy, and generalized lassitude and weakness. There was slight distention of the abdomen. Roentgenograms of the skull and chest were normal. Roentgenogram of the abdomen showed findings compatible with a moderate intestinal ileus. Electroencephalogram revealed a diffusely abnormal tracing with the most marked disturbance in the right central and occipital areas. All other laboratory studies were within normal limits, including urinary chloride excretion, except the serum chloride, which was 214 mg. per cent (340-350 mg. per cent being normal for the hospital laboratory).

The carbon dioxide combining power was 45.3 vol. per cent. It was decided that the abnormal serum chloride value was a laboratory error, but on the second hospital day repeat study showed serum chloride of 185 mg. per cent with again a normal CO_2 combining power. It was then that porphyria was suspected because of the low serum chloride in addition to the remainder of the clinical picture, and urine studies proved the diagnosis of porphyria which was later decided to be probably of toxic type.

This patient also had a moderate hypertension of 154/76 with a tachcardia of 120 beats per minute during her hospital stay.

The patient is now asymptomatic four months after leaving the hospital.

The above 2 case summaries are cited to point out the simulation of various surgical conditions, particularly those of the abdomen, by acute porphyria. In the 1 case a neurosurgical condition was suspected by the referring physician.

These cases are typical in that both underwent laparotomy prior to having the diagnosis of porphyria made. Frequently it may be impossible to rule out an acute condition within the abdomen by any means. There is no reason why both porphyria and a surgical condition of the abdomen cannot coexist, so undoubtedly it is wisest to explore surgically if the diagnosis of porphyria, once considered, cannot be substantiated. However, the principal point is that if the condition of porphyria is considered in the differential diagnosis of acute abdominal emergencies the number of needless explorations will be decreased.

SUMMARY

1. Recent literature on porphyria has been reviewed and presentation of the various aspects of porphyria has been made.

2. Inclusion of acute porphyria in the differential diagnosis of abdominal surgical disease has been emphasized.

3. Two case summaries illustrating this problem in diagnosis are presented.

REFERENCES

1. Abbott, K. H., and Evans, H. S.: Acute porphyria, Bull. Los Angeles Neurol. Soc. *11:*20 (March-June) 1946.
2. Abraham, A.: Gavey, C. J., and Maclagan, N. F.: Fatal case of acute porphyria with unusual features, Brit. M. J. *2:*327 (Aug. 30) 1947.
3. Aldrich, R. A., and others: Identification and crystallization of porphyrins in blood of child with light-sensitive (congenital) porphyria, Am. J. Dis. Child. *79:*1125 (June)1950.
4. Aldrich, R. A.; Hawkinson, V.; Grinstein, M., and Watson, C. J.: Photosensitive or congenital porphyria with hemolytic anemia. I. Clinical and fundamental studies before and after splenectomy, Blood *6:*685 (Aug.) 1951.
5. Aring, C. D., ed.: Neurological Clinical Pathological Conference of Cincinnati General Hospital, Case no. CH A-61321, Dis. Nerv. System *11:*247 (Aug.) 1950.

6. Ashby, D. W., and Bulmer, E.: Acute idiopathic porphyria, Brit. M. J. *2:*248 (July 29) 1950.
7. Avery, L. W.: Porphyria in relation to nervous and mental diseases, Dis. Nerv. System *6:*217 (July) 1945.
8. Baker, A. B., and Watson, C. J.: Central nervous system in porphyria, J. Neuropath. & Exper. Neurol. *4:*68 (Jan.) 1945.
9. Berg, M.: Acute porphyria, Arch. Int. Med. *76:*335 (Nov.-Dec.) 1945.
10. Berlin, L., and Cotton, R.: Gastrointestinal manifestations of porphyria, Am. J. Digest. Dis. *17:*110 (April) 1950.
11. Bittner, J. J., and Watson, C. J.: Possible association between porphyrins and cancer in mice, Cancer Research *6:*337 (July) 1946.
12. Brunsting, L. A., and Mason, H. L.: Porphyria with cutaneous manifestations, Arch. Dermat. & Syph. *60:*66 (July) 1949.
13. Brunsting, L. A., and Mason, H. L.: Porphyria with cutaneous manifestations, Proc. Staff. Meet., Mayo Clin. *22:*489 (Oct. 29) 1947.
14. Brunsting, L. A., and Mason, H. L.: Porphyria with epidermolysis bullosa; report of case of tardive congenital type with demonstration of latent porphyria in sister of patient, J.A.M.A. *132:*509 (Nov. 2) 1946.
15. Brunsting, L. A.; Mason, H. L., and Aldrich, R. A.: Adult form of chronic porphyria with cutaneous manifestations, J.A.M.A. *146:*1207 (July 28) 1951.
16. Calvy, G. L.: Porphyria, consideration in surgical diagnosis, Surg., Gynec. & Obst. *90:*716 (June) 1950.
17. Calvy, G. L.: Jaruszewski, E. J., and Carroll, H. H.: Porphyria; clinical observation and family vignette, Ann. Int. Med. *34:*767 (March) 1951.
18. Chandler, F. G.; Harrison, C. A., and Rimington, C.: Clinical porphyrinuria, Brit. M. J. *2:*1173 (Dec. 16) 1939.
19. Clark, E. E., and Lawrence, H. E.: Acute porphyria; report of case, J. Nerv. & Ment. Dis. *108:*502 (Dec.) 1948.
20. Coffman, D. O., and Kuhl, H. L.: Acute intermittent porphyria, U. S. Armed Forces M. J. *2:*315 (Feb.) 1951.
21. Coleman, R. R.: Acute porphyria, J. South Carolina M. A. *44:*117 (April) 1948.
22. Correll, H. L.: Peters, B. J., and Murphy, F. D.: Porphyria; report of two cases, Urol. & Cutan. Rev. *46:*341 (June) 1942.
23. Curtis, V. J., and Giliberti, J. J.: Acute porphyria in Negro, New York State J. Med. *50:*1966 (Aug. 15) 1950.
24. Denny-Brown, D., and Sciarra, D.: Changes in nervous system in acute porphyria, Brain *68:*1 (March) 1945.
25. Derrick, E. H.: Acute prophyria with reports of two fatal cases, M. J. Australia *1:*241 (Feb. 23) 1946.
26. Discombe, G., and D'Silva, J. L.: Acute idiopathic porphyria, Brit. M. J. *2:*491 (Oct. 13) 1945.
27. Dobriner, K., and Rhoads, C. P.: Porphyrins in health and disease, Physiol. Rev. *20:*416 (July) 1940.
28. Dobriner, K.: Strain, W. H.; Guild, H., and Localio, S. A.: Excretion of porphyrins in congenital porphyria, J. Clin. Ivestigation *17:*761 (Nov.) 1938.
29. Dunsky, I.: Freeman, S., and Gibson, S.: Porphyria and porphyrinuria, Am. J. Dis. Child. *74:*305 (Sept.) 1947.
30. Eliaser, M., Jr., and Kondo, B. O.: Electrocardiographic changes associated with acute porphyria, Am. Heart J. *24:*696 (Nov.) 1942.
31. Eriksen, L.: Acute porphyria. I. Some observations on solubility, precipitation, fluorescence and absorption spectra of calcium phosphate absorbed porphyrin pigments, Scandinav. J. Clin. & Lab. Invest. *3:*121, 1951.
32. Eriksen, L.: Acute porphyria. II. Some observations on conversion products from porphobilinogen, Scandinav. J. Clin. & Lab. Invest. *3:*128, 1951.
33. Eriksen, L.: Acute porphyria. III. Separation of preformed porphyrin and porphobilinogen in urine in acute porphyria, a coproporphyrin chromogen in normal and pathological urine, Scandinav. J. Clin. & Lab. Invest. *3:*135, 1951.
34. Figge, F. H. J.: Porphyrin metabolism: harderian gland fluorescence to susceptibility to carcinogenic agents, Cancer Research *4:*465 (Aug.) 1944.

35. Findlay, G. H., and Barnes, H. D.: Congenital porphyria, hydroa aestivale and hypertrichosis in South African Bantu, Lancet *2:*846 (Dec. 23) 1950.
36. Ford, W. H., and Ulrich, H. L.: Acute porphyria, Minnesota Med. *24:*254 (April) 1941.
37. Friedman, M. H.: Acute porphyria; case report, Bull. Georgetown Univ. Medical Center *1:*120 (Oct.-Nov.) 1947.
38. Garrod, A.: Congenital porphyrinuria; a postscript, Quart. J. Med. *5:*473 (Oct.) 1936.
39. Gibson, Q. H., and Harrison, D. C.: Note on urinary uroporphyrin in acute porphyria, Biochem. J. *46:*154 (Feb.) 1950.
40. Gibson, Q. H.; Harrison, D. C., and Montgomery, D. A.: Case of acute porphyria, Brit. M. J. *1:*275 (Feb. 4) 1950.
41. Golden, L. A.: Hematoporphyrinuric neuritis; report of case, Am. J. M. Sc. *206:*474 (Oct.) 1943.
42. Goldman, A. M., and Kaplan, M. H.: Acute porphyria, Ann. Int. Med. *34:*415 (Feb.) 1951.
43. Granick, S.: Iron and porphyrin metabolism in relation to red blood cell, Ann. New York Acad. Sc. *48:*657 (July 3) 1947.
44. Gray, C. H.: Acute porphyria; report of case, Arch. Int. Med. *85:*459 (March) 1950.
45. Gray, C. H.; Muir, I. M. H., and Neuberger, A.: Studies in congenital porphyria; incorporation of ^{15}N into haem and glycine of hemoglobin, Biochem. J. *47:*542 (Nov.-Dec.) 1950.
46. Gray, C. H., and Neuberger, A.: Studies in congenital porphyria. 1. Incorporation of ^{15}N into coproporphyrin, uroporphyrin and hippuric acid, Biochem. J. *47:*81 (June-July) 1951.
47. Gray, C. H.; Neuberger, A., and Sneath, P. H. A.: Studies in congenital porphyria. 2. Incorporation of ^{15}N in stercobilin in the normal and in the porphyric, Biochem. J. *47:*87 (June-July) 1950.
48. Gray, C. H.; Rimington, C., and Thomson, S.: Case of chronic porphyria associated with recurrent jaundice, Quart. J. Med. *17:*123 (April) 1948.
49. Grinstein, M., and others: Photosensitivity or congenital porphyria with hemolytic anemia. II. Isotopic studies of porphyria and hemoglobin metabolism, Blood *6:*699 (Aug.) 1951.
50. Grinstein, M.; Schwartz, S., and Watson, C. J.: Studies of uroporphyrins. I. Purification of uroporphyrin I and nature of Waldenstrom's uroporphyrin, as isolated from porphyria material, J. Biol. Chem *157:*323 (Jan.) 1945.
51. Grossfeld, E.: Acute porphyria with unusual features, Brit. M. J. *1:*1240 (June 2) 1951.
52. Grubschmidt, H. A.: Case of acute porphyria; remissions induced with procaine intravenously, California Med. *72:*243 (April) 1950.
53. Hall, W.: Acute intermittent porphyria; case, Minnesota Med. Foundation *4:*20 (Dec.) 1943.
54. Halpern, R. M., and Copsey, H. G.: Acute idiopathic porphyria, M. Clin. North America *30:*385 (March) 1946.
55. Hammond, R. L., and Welcker, M. L.: Porphobilinogen; tests on one thousand miscellaneous patients in search for false positive reactions, J. Lab. & Clin. Med. *33:*1254 (Oct.) 1948.
56. Hare, L., and Wilmor, R.: Acute porphyria, Am. J. Med. *5:*53 (July) 1948.
57. Hart, F. D., and Collard, P.: Acute idiopathic porphyria presenting as progressive paresis of Landry type, Brit. M. J. *1:*278 (Feb. 4) 1950.
58. Hayman, J. M., Jr.: Porphyria, Ohio State M. J. *45:*800 (Aug.) 1949.
59. Hetzel, B. S.: Acute porphyria, with report of two cases, M. J. Australia *2:*600 (Oct. 22) 1949.
60. Hoagland, P. I.: Acute porphyria, report of two cases with neurologic manifestations, Proc. Staff Meet., Mayo Clin. *17:*273 (May 6) 1942.
61. Hueper, W. C., and Figge, F. H. J.: Porphyrin excretion of harderian glands in its relation to actinic carcinogens in hairless rats, Cancer Research *5:*328 (June) 1945.

62. Jaffe, N. S.: Acute porphyria, associated with retinal hemorrhages and bilateral oculomotor nerve palsy, Am. J. Ophth *33:*470 (March) 1950.

63. Jorgensen, J., and With, T. K.: Acute porphyria, Lancet *1:*54 (Jan. 11) 1947.

64. Kiloh, L. G., and Nevin, S.: Acute porphyria with severe neurological changes, Proc. Roy. Soc. Med. *43:*948 (Dec.) 1950.

65. Kluver, H.: On naturally occurring porphyrins in central nervous system, Science *99:*482 (June 16) 1944.

66. Levy, S., and Klein, R. J.: Hematoporphyrin; review of literature and report of case of chronic type, Ann. Int. Med. *25:*520 (Sept.) 1946.

67. Linas, S.: Solomon, M. L., and Figge, F. H. J.: Chronic porphyria complicated by pregnancy, J.A.M.A. *133:*105 (Jan. 11) 1947.

68. Linder, G. C.: Salt metabolism in acute porphyria; report of two cases, Lancet *2:*649 (Nov. 1) 1947.

69. Little, N., and Palmer, H.: Acute porphyria; case report with notes on four others, New Zealand M. J. *47:*461 (Oct.) 1948.

70. Little, S. C., and Rose, F. E.: Acute porphyria; report of two cases, South. M. J. *38:*737 (Nov.) 1945.

71. Lowry, P., and Hawkinson, V.: Study of porphyrin and hemoglobin metabolism with aid of glycine-containing N^{15} in case of chronic (mixed) porphyria, J. Clin. Investigation. *29:*831 (June) 1950.

72. Lyons, H. A.: Acute porphyria with spinal fluid changes, Ann. Int. Med. *33:*711 (Sept.) 1950.

73. Marietta, J. S.: Porphyria, South. M. J. *42:*958 (Nov.) 1949.

74. McSwiney, R. R.: Nicholas, R. E. H., and Prunty, F. T. G.: Porphyrins of acute porphyria; detection of hitherto unrecognized porphyrins, Biochem. J. *46:*147 (Feb.) 1950.

75. Michaels, J. P.: Acute idiopathic porphyria; report of case, South. M. J. *42:*965 (Nov.) 1949.

76. Myerson, R. M.: Porphyria, Delaware State M. J. *23:*62 (March) 1951.

77. Nesbitt, S., and Watkins, C. H.: Acute porphyria, Am. J. M. Sc. *203:*74 (Jan.) 1942.

78. Neuberger, A.; Muir, H. M., and Gray, C. M.: Biosynthesis of porphyrin and congenital porphyria; Nature *165:*948 (June 17) 1950.

79. O'Leary, P. A. Montgomery, H.; Brunsting, L. A., and Kierland, R. R.: Chronic porphyria with cutaneous manifestations, Minnesota Dermatologic Soc. Case Presentations Sept. 9, 1949, Arch. Dermat. & Syph. *62:*596 (Oct.) 1950.

80. Palmer, H. W.: Case of acute idiopathic hematoporphyria with acute ascending paralysis, Ann. Int. Med. *13:*1500 (Feb.) 1940.

81. Peachey, C. H.: Dobriner, K., and Strain, W. H.: Hydroa estivale in congenital porphyria, New York State J. Med. *38:*848 (June 1) 1938.

82. Peters, G. A.: Acute porphyria; report of two cases with electrical studies in one, Ann. Int. Med. *30:*1237 (June) 1949.

83. Petrie, E.: Case of acute porphyria, Brit. M. J. *1:*926 (May 15) 1948.

84. Pohl, A. W., and Roberts, J. R.: Chronic porphyria; case report, Ann. Int. Med. *27:*1028 (Dec.) 1947.

85. Prunty, F. T. G.: Sodium and chloride depletion in acute porphyria with reference to status of adrenal cortical function, J. Clin. Investigation 690 (July) 1949.

86. Rau, L.: Acute idiopathic porphyria, Lancet *2:*647 (Nov. 23) 1940.

87. Sacks, C. L.: Porphyria, Hahneman. Month. *82:*310 (Aug.) 1947.

88. Schneck, J. M.: Porphyria; neuropsychiatric aspects in case of a Negro, J. Nerv. & Ment. Dis. *104:*432 (Oct.) 1946.

89. Schwartz, S., and Watson, C. J.: Isolation of uroporphyrin from feces in idiopathic porphyria, Proc. Soc. Exper. Biol. & Med. *47:*390 (May-June) 1941.

90. Scobey, R. R.: Porphyria and poliomyelitis, Arch. Pediat. *65:*131 (March) 1948.

91. Slade, C.: Porphyria, Canad. M. J. *60:*392 (April) 1949.

92. Sterling, K.: Silver, M., and Ricketts, H. T.: Development of porphyria in diabetes mellitus, Arch. Int. Med. *84:*965 (Dec.) 1949.

93. Strong, L. C., and Figge, F. H. J.: Fluorescence of harderian gland in mice of cancer susceptible and cancer resistant strains, Science *94:*331 (Oct. 3) 1941.
94. Taylor, F. R.: Porphyria, Urol. & Cutan. Rev. *46:*104 (Feb.) 1942.
95. Taylor, I. J.: Solomon, M. L.: Weiland, G. S., and Figge, F. H. J.: Chronic porphyria, J.A.M.A. *131:*26 (May 4) 1946.
96. Turner, W. J.: Studies on porphyrin. III. Acute idiopathic porphyria, Arch. Int. Med. *61:*762 (May) 1938.
97. Watson, C. J.: Porphyria, South. M. J. *36:*359 (May) 1943.
98. Watson, C. J.: Porphyrin metabolism and porphyria, Brit. M. J. *2:*574 (Sept. 2) 1950.
99. Watson, C. J., and Schwartz, S.: Simple test for urinary porphobilinogen, Proc. Soc. Exper. Biol. & Med. *47:*393 (May-June) 1941.
100. Watson, C. J.; Schwartz, S., and Hawkinson, V.: Studies of uroporphyrins. II. Further studies of porphyrins of urine, feces, bile and liver in cases of porphyria, with particular reference to Waldenstrom type porphyrin behaving as entity on Tswett column, J. Biol. Chem. *157:*345 (Jan.) 1945.
101. Welcker, M. L.; Porphyrins, New England J. Med. *232:*11 (Jan. 4) 1945.
102. Wiggins, C. A.: Fatal case of acute porphyria in Negro, Brit. M. J. *2:*866 (Oct. 14) 1950.
103. Woody, N. C.: Porphyrinuria; report of unusual case, Pediatrics, *4:*47 (July) 1949.
104. Yaeger, C. L.: Polyneuritis; differentiation of infectious polyneuritis (Guillain-Barre syndrome) and neuritis of porphyria, Minnesota Med. *30:*166 (Feb.) 1947.
105. Zeligman, I.: Red fluorescence of urine in Wood's light as aid in office diagnosis of porphyria, Arch. Dermat. & Syph. *61:*853 (May) 1950.

OBTURATOR HERNIA

Ed. B. Rowe, M.D.
Gustavus A. Bynum, M.D.
Galveston

Although obturator hernia is uncommon, it is nevertheless an important disorder due to the serious consequences which develop if treatment is not prompt. It is also of interest because the diagnosis is seldom made before laparotomy, and even at laparotomy the condition may be overlooked if the possibility of its occurrence is not realized. It is probable that the incidence of correct preoperative diagnosis of obturator hernia will increase as knowledge of the condition becomes more generalized.

In 1724 Arnaud De Ronsil gave the first report of a case of obturator hernia before the Academy of Surgeons in Paris. In 1743 Garengeot read a paper on obturator hernia before the Royal Academy of Surgery in Paris, reporting a case of his own in 1733, and 6 others collected from the literature. Camper described the anatomy of this hernia in 1762. In 1768 a case was reported by G. Arnaud, son of Arnaud De Ronsil, and this was the first instance in which a strangulated obturator hernia was reduced successfully by taxis. Laparotomy for strangulated obturator hernia was first performed by Hilton in 1848, Coulson in 1863, and Godlee in 1885. Their patients died. According to Erickson in *Lancet,* 1850, the first case successfully detected and operated upon with recovery was by Henry Obre. After the beginning of antiseptic surgery more and more cases were cured by operation.

In 1946 Watson collected 442 cases of obturator hernia from the literature. There have been 12 cases recorded since 1946, and these along with 1 reported in this paper, make a total of 455 cases. These figures are probably inaccurate and serve only as a rough estimate of the infrequency of this condition.

A knowledge of the anatomy of the obturator region is necessary to appreciate the origin, clinical course, and treatment of obturator hernia. The location of the herniation is the obturator foramen and canal in the innominate bone which is on each anterolateral surface of the pelvis. The obturator foramen is closed by the tough obturator membrane, which is deficient in its superior portion to allow for the passage of the obturator vessels and nerve from the pelvis into the thigh. The obturator membrane is reinforced internally by the obturator internus muscle and externally by the

From the Department of Surgery, University of Texas School of Medicine, Galveston, Texas.

obturator externus muscle. Both of these muscles take partial origin from the obturator membrane, and are powerful lateral rotators of the thigh. The obturator canal transmits the obturator nerve and vessels. The canal runs obliquely downward and forward and is about 2 to 3 cm. in length.

The pelvic peritoneum covers the internal opening of the obturator canal. Immediately under the peritoneum is a layer of fatty tissue which to a certain extent prevents herniation of intra-abdominal organs into the canal; however, fat may prolapse into the canal and give rise to a fatty hernia. In the region of the obturator canal the preperitoneal fatty layer is an extension of the bladder fat and is moderately thick and somewhat tough. The parietal portion of the endopelvic fascia, against which the preperitoneal fat rests, is the covering of the obturator internus muscle, and this fascia is carried into the obturator canal and fuses with the periosteum and the tissue of the obturator membrane. The internal obturator opening is surrounded by very dense and unyielding structures. The external obturator opening is beneath the pectineus, adductor longus, and obturator externus muscles.

The obturator nerve supplies the skin, muscles, and fascia over the medial part of the thigh. It also gives a branch to the hip joint and one to the posterior part of the knee joint. The obturator artery has the same general distribution. The nerve and vessels are found in the fatty layer beneath the peritoneum.

It seems, then, that the obturator region is well protected by strong, dense structures, and this is undoubtedly one of the chief reasons why hernia is rare at this location. Also this anatomic arrangement explains why obturator hernia is accompanied by strangulation in a high percentage of cases. A study of the anatomy shows the futility of trying to repair an obturator hernia by exposure through the thigh. One is interested in closing the internal opening so intra-abdominal contents will not enter the canal.

The sac of an obturator hernia is formed by peritoneum which is forced downward into the canal by increased intra-abdominal pressure. These hernias are usually small due to the size of the canal and the strength of the surrounding structures. Small intestine is the most common content of the sac, and in those cases which are incarcerated or strangulated it is common for the obstruction to be of the Richter's type in which only a portion of the bowel circumference is involved.

The relationship of the neck of the sac to the position of the obturator vessels and nerve is somewhat variable. In the case of the artery, this variability is explained partly by different positions

of the sac and partly by the fact that the obturator artery sometimes has an anomalous origin from the deep epigastric artery. In most cases, the obturator artery is behind and lateral to the neck of the hernial sac. The nerve is usually on the lateral side of the sac and above the artery; however, it may be in front of the sac, and rarely, behind it or to the medial side.

Obturator hernia occurs about five or six times more frequently in women than in men. There are several factors which tend to explain this occurrence. The obturator foramen is consistently larger in women than in men. Frequent pregnancies cause a relaxation of the pelvic peritoneum and also tend to cause a thinning of the protective preperitoneal fatty layer. Emaciation may remove this protective fatty layer. About 10 to 15 per cent of obturator hernias occur in males. By far the majority of these hernias occur in the 60 to 70 year age group, and it is rare to see one in a patient under 50 years of age. Chronic pulmonary diseases and chronic constipation are predisposing causes due to the increased intra-abdominal pressure which these conditions produce. An extraperitoneal lipoma in the region of the obturator foramen may pull the peritoneum into the canal and so initiate the formation of a hernia.

In those cases not complicated by incarceration or strangulation, the symptoms and signs will usually not be specific enough to allow a correct diagnosis to be made unless there is a palpable or reducible swelling present in the thigh over the area of the external obturator opening. It is rare for an obturator hernia to be unaccompanied by incarceration or strangulation. The point of constriction is nearly always at the neck of the sac at the internal opening. Even in incarcerated or strangulated obturator hernias, the correct diagnosis will seldom be made before operation unless the patient exhibits certain signs and symptoms which are diagnostic.

The patient will usually give a history of a sudden onset of cramping abdominal pain which may be generalized or localized to the right or left lower abdomen. This pain will be accompanied by nausea and vomiting. These are the symptoms of acute intestinal obstruction. Some of these patients will give a history of similar episodes in the past which subsided spontaneously. Pain along the course of the obturator nerve is the important symptom suggesting obturator hernia. This pain is probably due to pressure on the nerve by the hernial sac and its contents, and it is known as the Howship-Romberg sign. This sign is present in about half of the cases. Because of pain, the patient may complain on motion of the thigh or hip, and he may keep his thigh flexed so as to relieve the stretch on the nerve.

On examination of the patient all the signs of intestinal obstruction may be found, including profuse vomiting, abdominal distention, dehydration, and high pitched peristalsis. In addition there may be localized tenderness in one lower quadrant of the abdomen with localized muscle guarding or spasm. The typical Howship-Romberg sign may be present, and any movement of the hip or thigh may cause severe pain. There may be a detectable bulge over the upper medial part of the thigh in the external obturator region. The presence of such a swelling associated with tenderness is an important clue to the diagnosis. By vaginal or rectal examination a tender mass on the anterolateral surface of the pelvis at the location of the internal obturator foramen may be felt.

Those patients coming to the hospital late will be desperately ill so that a specific diagnosis is difficult to make. Even in those who are seen early the incidence of correct preoperative diagnosis will be relatively low. The important fact is that it makes little difference whether or not a correct preoperative diagnosis is made, just so the condition is recognized and treated as an acute condition in the abdomen requiring surgery or as an acute intestinal obstruction. A flat and upright roentgenogram of the abdomen will usually serve to differentiate between large bowel obstruction and small bowel obstruction, and will differentiate between high and low small bowel obstruction. The proper abdominal incision can be made accordingly. At operation the cause of the obstruction can be found and the condition relieved.

Incarcerated obturator hernia will have to be differentiated from all conditions which produce acute intestinal obstruction. Incarcerated right obturator hernia may be erroneously diagnosed as acute appendicitis, especially if seen early before the signs of intestinal obstruction have become apparent; it is very important not to overlook the condition at operation in such a case. Many other conditions may be confused with obturator hernia; however, the following are the most important: femoral hernia, femoral adenitis, pelvic inflammatory disease, ruptured ectopic pregnancy, hip joint disease, and diverticulitis of the sigmoid.

The prognosis of obturator hernia is dependent on the condition of the patient, the duration of the illness, and the presence of strangulation. The mortality is somewhat high for several reasons: (1) the condition is associated with a high percentage of incarceration and strangulation; (2) the patients are commonly old and debilitated; (3) an incorrect diagnosis is frequently made and improper treatment instituted. If all cases of incarcerated or strangulated obturator hernia were managed as cases of acute intestinal obstruction the mortality rate would decline.

The treatment of this disorder is entirely a surgical problem. Those hernias which are diagnosed before incarceration should be repaired as soon as possible because of the great tendency to incarceration. In our opinion, trusses have no place in the treatment of obturator hernias; an external truss could not keep the hernia from entering the internal obturator foramen, whereas the entrance is the most frequent site of constriction.

Most of the cases are incarcerated or strangulated when first seen. Those few cases in which a palpable or visible swelling is present in the external obturator region should not be subjected to prolonged attempts at reduction. Due to the inelasticity of the surrounding structures, any attempt at reduction may produce damage to the contents of the sac with possible disastrous results.

Several different operative procedures have been developed for the treatment of obturator hernias. An approach may be made through the thigh, and the sac located and treated. This method is unpopular at present due to the difficulty of the exposure. If necrotic bowel is present, the resection is quite difficult through such a small opening, and in many cases it is necessary to open the abdomen to accomplish this. Also the vessels are difficult to identify through the thigh and thus are more apt to be injured with resulting hemorrhage. To us this route is unsatisfactory since it does not allow the internal obturator foramen to be closed securely. If the internal obturator foramen is not closed recurrence is likely.

The inguinal approach is somewhat better than the approach through the thigh, but still does not seem adequate for treatment of incarcerated or strangulated hernias. In the case of a Richter's type of hernia, manipulation through an inguinal incision may cause reduction of the hernia, and the abdomen will then have to be opened to determine the viability of the bowel involved.

The abdominal route appears for several reasons to be the method of choice for dealing with obturator hernias. The majority will be diagnosed correctly only at laparotomy, and fortunately such a hernia can best be repaired transabdominally. The type of abdominal incision is a matter of personal preference; however, we were able to obtain adequate exposure in our case by a lower abdominal transverse incision. Upon entering the abdomen, the hernial orifice is usually easily found, and in most cases reduction of the sac contents is relatively simple. The viability of the bowel can be determined, and resection carried out if necessary. A few patients will be in such poor general condition at the time of operation that it is possibly justifiable to reduce the hernia and close the abdomen without making any repair of the hernial defect. Preferably a repair of

the internal opening should be done to prevent recurrence.

The simplest and quickest treatment of the sac is inversion of the peritoneum forming the sac and ligation of the neck. The excess sac may then be trimmed away, or, better, it may be quilted over the foramen to reinforce the repair. Wakeley has advocated ligation of the sac and suture of a strip of the innermost fibers of the pectineus muscle, raised through an incision in the groin, to the periosteum above the obturator canal. Short (1923) used a piece of rib cartilage to plug the obturator canal. This would seem to be a good method of repair, but the possibility of causing obturator neuralgia by such a procedure should be considered. An occasional case has been reported in which it was necessary to make both an abdominal incision and a thigh incision to accomplish reduction of the sac contents.

Case Report

F. M., a 65 year old white man, was first seen at the John Sealy Hospital on Nov. 19, 1951, at which time he complained of severe, generalized, cramping abdominal pain. Eight days previously he had a sudden onset of severe pain in the right hip and right inguinal region. This pain also radiated into the penis. He was seen by a physician who made a diagnosis of kidney stone, and gave him a hypodermic and oral medication. In the following 24 hours the patient had a rather severe diarrhea with an increase in severity of the abdominal cramps. Thereafter the patient had passed no stool or gas per rectum, and the abdomen had become progressively more distended. Since the second day of the illness vomiting had been frequent. There was no history of any previous similar illness. At the time the patient was first seen by us he had no hip pain or leg pain. There was a history of chronic productive cough of about 10 years' duration.

Physical examination showed temperature 98.6 F., pulse 120, respiration 26, and blood pressure 108/92. The patient was somewhat emaciated, very dehydrated, and acutely ill. The respirations were rapid and labored, although no cyanosis was present. The abdomen was quite distended and dilated loops of bowel could be seen through the abdominal wall. There was generalized abdominal tenderness, more severe in the right lower quadrant, although no rigidity or muscle guarding was present. Bilateral inguinal hernias were present and were easily reduced with no pain. No bulges were noticed in the upper thighs, and the patient complained of no pain on movement of the legs. Auscultation of the abdomen revealed high pitched obstructive type of peristalsis. Rectal examination showed the ampulla to be empty; however, a large loop of bowel could be felt in the pelvis, but it was not particularly tender.

A chest roentgenogram was made, and showed extensive tuberculosis in the upper lung field on the right with multiple cavities. Flat and upright roentgenograms of the abdomen showed many dilated loops of small bowel with multiple air-fluid levels.

A diagnosis of mechanical obstruction of the terminal ileum was made. Intestinal decompression was begun by means of a Miller-Abbott tube, antibiotics were administered, and intravenous fluids given. The patient responded

well to these measures, and 12 hours after admission he was taken to the operating room where the abdomen was opened through a right lower quadrant transverse incision. An incarcerated right obturator hernia was found. The hernia was of the Richter's type involving the terminal ileum about 3 feet from the ileocecal valve. The incarceration was reduced and the bowel was viable. A circular incision through the peritoneum was made at the internal obturator foramen, thus severing the hernial sac. The peritoneum lining the sac was dissected out and discarded. The parietal preitoneum was dissected up for a distance of 1½ inches surrounding the internal obturator foramen. The preperitoneal fat was reflected from the obturator internus muscle and the layer of fascia covering the obturator internus muscle was exposed. In the process of this dissection, the vessels and nerve were identified and avoided. The fascia over the obturator internus muscle was then sutured securely across the internal obturator foramen to close it. The preperitoneal fat and peritoneum were closed. All sutures were of No. 000 braided silk. The appendix was removed and the abdomen was closed. By the fourth postoperative day the patient was able to take fluids by mouth. At the end of a week the wound was well healed; the patient was discharged home on the ninth postoperative day at which time he had no abdominal complaints.

The method described seemed to give a very adequate repair. It is simple to perform, rapid, and the materials for repair are close at hand.

Summary

A review of the pertinent facts concerning obturator hernia is given. The following points are helpful in making a diagnosis of incarcerated obturator hernia: (1) acute intestinal obstruction associated with a positive Howship-Romberg sign; (2) palpation of a tender mass on the lateral pelvic wall by vaginal or rectal examination; (3) palpation of a lump or tenderness in Scarpa's triangle; and (4) partial flexion of the hip, and exacerbation of the pain by movements of the hip.

Incarcerated obturator hernias should be treated as cases of acute intestinal obstruction. One case of incarcerated obturator hernia is presented, and a method of repair is suggested.

REFERENCES

1. Adams, H. D., and Smith, D. C.: Obturator hernia; report of successful operative case, J.A.M.A. *137:*948 (July 10) 1948.
2. Anson, B. J.; McCormack, L. J., and Cleveland, H. C.: Anatomy of hernial regions; obturator hernia and general considerations, Surg., Gynec. & Obst. *90:*31 (Jan.) 1950.
3. Desmond, A. M., and Hutter, F.: Strangulated obturator hernia, Brit. J. Surg. *35:*318 (Jan.) 1948.
4. Franklin, R. H.: Obturator hernia, Lancet *1:*721 (March 26) 1938.
5. Horine, C. F.: Obturator hernia, Ann. Surg. *86:*776 (Nov.) 1927.
6. Iason, A. H.: Hernia, Philadelphia, The Blakiston Company, 1941
7. Pernworth, P.: Obturator hernia; report of operation for irreducible incarceration, Am. J. Surg. *71:*539 (April) 1946.
8. Rose, T. F.: Obturator hernia; case report, M. J. Australia *2:*260 (Aug. 12) 1950.

9. Rothman, M.: Repair of obturator hernia with peritoneal graft, New York J. Med. *51:*1186 (May 1) 1951.
10. Saleeby, E.: Obturator hernia; report of successful operation in eighty-three year old woman, Am. J. Surg. *65:*422 (Sept.) 1944.
11. Van Zwalenburg, C.: Final report on case of radical operation for cure of double obturator hernia; failure, Surg., Gynec. & Obst. *33:*429 (Oct.) 1921.
12. Wakeley, C. P. G.: Obturator hernia; its etiology, incidence and treatment, with two personal operative cases, Brit. J. Surg. *26:*515 (Jan.) 1939.
13. Waterfall, M. C.: Recovery from strangulated obturator hernia, Brit. M. J. *1:*585 (March 11) 1950.
14. Watson, L. F.: Hernia, St. Louis, C. V. Mosby Company, 1948.
15. Yanover, R. R.; Podolsky, W. G., and Marks, C. R.: Obturator hernia, New York J. Med. *50:*2317 (Oct. 1) 1950.

THE IMPORTANCE OF SPECIFIC ANTIBIOTIC THERAPY IN SURGICAL INFECTIONS

CHARLES D. DUKES, Ph.D.
Galveston

DURING the past half decade the surgeon has received a potent addition to the armamentarium in the fight against surgical infections with the advent of the major antibiotics. The literature, coupled with circulars from the pharmaceutical houses, is well sprinkled with glowing reports of the efficacy and importance of the major antibiotics in almost every type of infection encountered by the surgeon.

Many reports stress the applicability of one antibiotic or another in the treatment of specific infectious entities. These reports frequently ignore the desirability of obtaining the drug of choice in each infection, the effect of the antibiotic on the bacterial flora present, and the constant possibility of the development of resistance of the infectious entity to any one antibiotic. Consequently, for those who have not been extensively schooled in the mechanism of infection and the scope of anti-infective agents, it is frequently difficult to select the broad facts and basic concepts of truth from the maze of information varying from excellent clinical and laboratory research to highly circumscribed observation and, in some cases, wishful thinking.

There are many excellent publications in the literature dealing with the background and major concepts of antibiotic therapy in the special field of surgical infection. Most of these papers sift out the misinformation while retaining the pertinent facts and are to be highly recommended for general reading.[1,2,13,17,18]

It is not within the scope of this paper to reiterate these basic facts but rather to voice a plea for specific therapy of all infections whenever possible, to discuss the growing problem of infection by multiple species of bacteria and to postulate a few guiding notes to deal with these problems.

DEVELOPMENT OF RESISTANCE

When penicillin was introduced by Fleming and Florey in 1940, the possibility of banishing staphylococcal infections from the surgical wards became one of the brightest dreams of the day. By 1948 it became quite evident that the staphylococci were capable of spontaneously developing resistance to penicillin during therapy and the

From the Department of Surgery, Plastic and Maxillofacial Division, University of Texas Medical Branch, Galveston.

bright dream began to fade. Now, in 1952, penicillin is frequently no longer the drug of choice in the case of staphylococci encountered in surgical infections. Over 60 per cent of the strains isolated in this laboratory are resistant to penicillin and the percentage is growing yearly.[5] A great deal of this development of resistance can be attributed to inadequate dosage, empirical application and general misuse of this highly important drug. The frequent tendency of physicians to prescribe "one-shot" treatments of penicillin for nonspecific infections is obviously to be deplored. This process is one of the greatest contributing factors in the recognized increasing incidence of resistance to penicillin in the case of the pyogenic cocci. The low blood levels of between 0.6 and 1.8 units per ml. produced by a single injection of procaine penicillin frequently is sufficient to provoke the well known step-wise development of increased resistance. When this process is repeated at varying intervals by nonspecific therapy, extremely resistant strains of pyogenic cocci can result. Inadequate specific therapy, either too low a dosage or too short a period of treatment, also contributes heavily to the development of resistance.

Unfortunately, the increasing incidence of resistance of bacteria which at one time were universally susceptible to the action of antibiotics is by no means restricted to penicillin nor to staphylococci. Similar trends are apparent for other bacteria and other antibiotics.[5,7,19]

Prophylactic Use of Antibiotics

With the introduction of the broad spectrum antibiotics such as aureomycin, chloromycetin, and terramycin, it was felt by many that these agents would provide more applicable prophylactic measures because of their wider range of antibacterial ability to combat infections against which penicillin had little or no effect. Unfortunately, these broad spectrum antibiotics have been infrequently considered in the selection of prophylactic agents and penicillin still remains the drug of choice in many parts of this country. This routine use of penicillin completely ignores the possibility of infection by Gram-negative bacilli and other resistant bacteria.

It is quite easy to defend the indiscriminate use of antibiotics as a prophylactic measure in *all* surgical procedures by the pious declaration that *all* patients deserve protection against infection. Yet in many cases, particularly in the clean, elective type of surgery not involving an infectious process or the alimentary canal, there is little excuse for infection, consequently little need for prophylactic precaution.

Prophylaxis *should* be employed by all means in such procedures

as those involving infectious processes, gastrointestinal and urinary tracts, burns, and contaminated wounds, and patients with diabetes, agranulocytosis, rheumatic heart disease, and valvular lesions, either as a protection for the patient or as an adjunct to the surgery itself. However, intelligence is needed to select those antibiotic or chemotherapeutic agents which will provide the greatest protection for the patient. Extensive research has been done on the sterilization of the intestinal tract[3,4,15,16] as well as repeated reports on the current comparative susceptibility of commonly encountered bacteria to the various antibiotics.[7,8,9,19,21] The conscientious surgeon makes use of these published data in the choice of the drug to be utilized as a prophylactic agent.

Specific Therapy

When infection is encountered in the surgical patient, it is of prime importance to obtain cultural and antibiotic sensitivity studies whenever feasible. Such information is often as important to the surgeon as proper roentgenologic or hematologic examination. Most hospitals in the United States are equipped and able to render such diagnostic service within a reasonable period of time and at a reasonable cost to the patient. Failure to utilize such facilities will frequently lead to the empirical use successively of from two to five antibiotics over a period of up to several weeks before the infection is brought under control. In a significant number of infections, the possible etiology may be deduced by direct microscopic examination of stained smears and the antibiotic most *likely* to be of assistance selected until such time as the cultures may be obtained and the antibiotic of choice determined specifically.

When the laboratory information regarding the relative sensitivity of the infectious agent is utilized in the treatment of the infection, time, expense, and complications may frequently be reduced to a minimum. Definitive knowledge of the average blood level attained with any given dosage schedule will often mean the difference between rapid elimination of the infection or the persistence of infection due to the development of resistant bacteria. Inadequate antibiotic therapy is one of the greatest contributing factors to the development of chronicity in infection and the increasing incidence of resistant bacteria.

At the present time, antibiotic sensitivity determinations are obtained by two main methods, the medicated disc and the tube dilution method. The tube dilution method is by far the more accurate and to be desired in general and particularly in the case of resistant infections. The disc method is by its nature the more rapid method

and should be utilized mainly in those infections which are critical and which require specific therapy in the shortest possible period of time. The disc method is useful as a screening procedure and will give approximate figures of sensitivity but it is fraught with modifying characteristics inherent in the method itself and therefore frequently difficult to evaluate properly.

Mixed Infection

One of the most complex problems arising in the field of surgical infection today is that of mixed bacterial infection particularly in surface lesions and subsequent to extensive surgical procedures involving contaminated or infected tissue.

The prominence of these mixed infections is due to a number of factors including the improper use of antibiotics, the development of resistance of bacteria and the alteration of bacterial flora during antibiotic therapy. Recent research has definitely established the fact that the use of antibiotics may bring about marked alterations in the bacterial flora in various parts of the body.[10,11,20,22,23] This alteration may be manifested in the area of infection by the inhibition of susceptible organisms and the accentuated growth of organisms present in small numbers which are not sensitive to the antibiotic. The flourishing of these resistant bacteria may result in the invasion of tissue and the production of a new infection. If several species are present, a potent mixed infection may develop of greater consequence than the one originally treated. While such instances are not common, they are by no means rare in their occurrence.

A large majority of the mixed infections involve one or more bacteria which are resistant to the action of penicillin either inherently or through the medium of developed resistance. One of the more frequently encountered mixtures in surface infections consists of penicillin-resistant *Staphylococcus aureus* and *Pseudomonas aeruginosa*. This combination is often very damaging to the patient from the standpoint of toxemia and destruction of tissue, particularly in the case of extensive burns, multiple lacerations, and topical ulcerations. Treatment with any single antibiotic is usually unrewarding; in our experience less than 20 per cent of such infections have responded to systemic therapy with any one of the broad spectrum antibiotics. Excellent results have been achieved in a series of 36 cases by the administration of aureomycin orally and aqueous solutions of polymyxin B topically.[6]

Altemeier and Culbertson were able to demonstrate good results in two thirds of mixed Gram-negative and Gram-positive surgical infections by the use of chloromycetin or aureomycin. However,

the initial favorable response was apt to be temporary if proper surgical treatment was not carried out concomitantly.[2] Another mixed infection which is potent in its destructive effects is that of *Staphylococcus aureus* and a nonhemolytic streptococcus associated in progressive bacterial synergistic gangrene. This condition has been extensively studied by Meleney[12] who has obtained excellent results with systemic bacitracin after the condition had failed to respond to penicillin.[14] However, it is generally of prime importance to determine the antibiotic sensitivity of the bacteria involved in mixed infections in order to attain maximum beneficial results from such therapy. It should be reemphasized that rarely will the antibiotics eliminate the necessity of proper surgical intervention in those conditions which ordinarily require such procedures.

Summary

The problem of surgical infections, which generally has been minimized during this antibiotic age, is still a serious factor worthy of intelligent consideration. A plea is voiced for specific antibiotic therapy based on the determination of the etiologic agents and upon their antibiotic sensitivity as demonstrated by *in vitro* methods whenever possible.

References

1. Altemeier, W. A.: Chemotherapy in Surgery, in Carter, B. N., ed.: Monographs on Surgery, New York, Thomas Nelson & Sons, 1950, chap. 1.
2. Altemeier, W. A., and Culbertson, W. R.: Chloramphenicol (chloromycetin) and aureomycin in surgical infections, J.A.M.A. *145:*449 (Feb. 17) 1951.
3. Baker, H. J., and Pulaski, E. J.: Effects of terramycin on fecal flora, Ann. New York Acad. Sci. *53:*324 (Sept. 15) 1950.
4. Bierman, H. R., and Jawetz, E.: Effect of prolonged administration of antibiotics on human fecal flora, J. Lab. & Clin. Med. *37:*394 (March) 1951.
5. Dukes, C. D.: Growing incidence of resistance of Staphylococcus aureus to major antibiotics (in press).
6. Dukes, C. D.: Unpublished data.
7. Frank, P. F.; Wilcox, C., and Finland, M.: In vitro sensitivity of coliform bacilli to seven antibiotics, J. Lab. & Clin. Med. *35:*188 (Feb.) 1950.
8. Frank, P. F.; Wilcox, C., and Finland, M.: In vitro sensitivity of Bacillus proteus and Pseudomonas aeruginosa to seven antibiotics, J. Lab. & Clin. Med. *35:*205 (Feb.) 1950.
9. Hamburger, M., and Muething, D.: In vitro sensitivity to eight antibiotics of streptococci and streptococci recovered from cases of bacterial endocarditis, J. Lab. & Clin. Med. *37:*60 (Jan.) 1951.
10. Keefer, C. S.: Alteration in normal bacterial flora of man and secondary infections during antibiotic therapy (Editorial) Am. J. Med. *11:*665 (Dec.) 1951.
11. Lipman, M. O.; Coss, J. A., and Boots, R. H.: Changes in bacterial flora of throat and intestinal tract during prolonged oral administration of penicillin, J. Bact. *51:*594 (May) 1946.
12. Meleney, F. L.: Differential diagnosis between certain types of infectious gangrene of skin, with particular reference to hemolytic streptococcus gangrene and bacterial synergistic gangrene, Surg., Gynec. & Obst. *56:*847 (May) 1933.

13. Meleney, F. L.: Intelligent use of antibiotics in prophylaxis and treatment of surgical infections, New York Med. *5:*15 (Dec.) 1949.
14. Meleney, F. L.; Shambaugh, P., and Millen, R. S.: Systemic bacitracin in treatment of progressive bacterial synergistic gangrene, Ann. Surg. *131:*129 (Feb.) 1950.
15. Poth, E. J.; Fromm, S. M.; Wise, R. I., and Hsiang, C. M.: Neomycin, new intestinal antiseptic, Texas Rep. Biol. & Med. *8:*353, 1950.
16. Pulaski, E. J.; Connell, J. F., Jr., and Seeley, S. F.: Sterilization of intestinal tract by antibiotics and supplemental agents, Ann. Surg. *132:*225 (Aug.) 1950.
17. Pulaski, E. J.: Place of antibiotics in field of surgery, Ann. New York Acad. Sci. *53:*347 (Sept. 15) 1950.
18. Pulaski, E. J., and Shaeffer, J. R.: Background of antibiotic therapy in surgical infections, Internat. Abstr. Surg. *93:*1, in Surg., Gynec. & Obst. (July) 1951.
19. Ransmeier, J. C.; Brown, H. E., and Davis, N.: Comparison of in vitro sensitivity of freshly isolated pathogenic bacteria to terramycin and other antibiotics, J. Lab. & Clin. Med. *38:*620 (Oct.) 1951.
20. Sommer, L. S., and Favour, C. B.: Biologic complications of penicillin therapy, Am. J. Med. *7:*511 (Oct.) 1949.
21. Spink, W. W.: Clinical and biologic significance of penicillin-resistant staphylococci including observations with streptomycin, aureomycin, chloramphenicol and terramycin, J. Lab. & Clin. Med. *37:*278 (Feb.) 1951.
22. Weinstein, L.: Spontaneous occurrence of new bacterial infection during course of treatment with streptomycin or penicillin, Am. J. Med. Sci. *214:*56 (July) 1947.
23. Woods, J. W.; Manning, I. H., Jr., and Patterson, C. N.: Monilial infections complicating therapeutic use of antibiotics, J.A.M.A. *145:*207 (Jan. 27) 1951.

MANAGEMENT OF THE URINARY BLADDER IN THE PARAPLEGIC

OLIVER H. GRAVES, M.D.*
Galveston

SPINAL cord injuries present an intriguing and controversial urologic problem. Surgeons most experienced in the handling of these patients are not in complete agreement on the best method of management. Because of immediate shock, it is impossible at first to determine exactly the extent of the injury or the degree of permanence. The neurosurgeon and urologist should work in close cooperation because the level and severity of the spinal injury determine the expected degree of bladder recovery. Most patients with partial transection of the cord will progress to a stage of voluntary micturition. Those with complete transection are likely to progress to a stage known as automatic bladder provided the lesion is above the conus. Lesions of the conus or cauda equina are more likely to result in an autonomous bladder.

Urologic care must be influenced by three objectives: (1) prevention of urinary tract infection; (2) maintenance of bladder capacity within normal limits; (3) recognition of the automatic stage of bladder recovery which may permit removal of the catheter and allow intermittent voiding.

Bladder capacity may be maintained within normal limits by manual expression, transurethral drainage, or suprapubic drainage. He who resorts to intermittent catheterization qualifies well as a "Lord High Executioner." No matter how careful his aseptic precautions are, he will lose most of his patients from urinary tract infection. So "spare the catheter and save the life" cannot be emphasized too strongly. These patients do not die of retention nor does retention of itself permanently injure the urinary tract.

It is preferable to have bladder paralysis demonstrate itself by doing nothing for 12 to 18 hours or even up to 24 hours if it requires that length of time for the bladder to fill. If urinary retention occurs, manual expression of the urine every four hours may be tried. If this fails, an F 18 Foley retention catheter is inserted. If this is not done, the bladder overflows and the patient stays wet from overflow incontinence. Being constantly bathed in urine, the skin over the sacrum is more likely to break down to form a decubitus ulcer. Munro has urged the use of tidal drainage, cystom-

From the Department of Surgery, Urological Division, University of Texas Medical Branch, Galveston.
From the Department of Surgery, University of Texas School of Medicine, Galveston, Texas.

etry, and bladder training in numerous publications and reports amazing results. By tidal drainage the bladder is alternately and mechanically filled and emptied. Puddling of residual urine and overstretching or shrinkage of the bladder wall are prevented, and the bladder is maintained in a close approximation to a physiologically normal state. Its proper use depends on cystometrographic demonstration of the type of bladder to be served depending on the stage of bladder recovery.

A cystometric determination shows the functional activity of the bladder and is simply and easily measured by instilling small increments of water into the bladder and reading the intravesical pressure in millimeters of mercury or cubic centimeters of water. The intravesical pressure is controlled by the level of the siphon which is set just below the intravesical pressure in centimeters produced by the amount of fluid which the bladder should contain, for example, 200 cc. For the atonic bladder the siphon is set 1 to 2 cm. above the bladder level. As the bladder becomes hypertonic, this is raised. For the reflex bladder about 10 to 12 cm. is the usual level.

It is important to emphasize that, for tidal drainage to be successful, attendants, including physicians, nurses, and ward personnel, must pay meticulous attention to minute details at all times.

In cases of complete transection of the cord above the conus, the bladder goes through four stages of recovery: (1) atonicity, (2) autonomicity, (3) hypertonicity, and (4) reflex automatic bladder. The graph of the atonic bladder is flat, that of the autonomous bladder shows weak ineffective contractions, while the automatic bladder shows strong effective contractions. The period of recovery may require 3 to 10 months, the average being about five months.

After the final stage of bladder recovery is established, a period of bladder training is essential. For this to succeed, the urinary tract must ideally be free of infection, relatively unobstructed, and preferably free of stones. The patient must be in a state of good nutrition, cooperative, intelligent, and, if possible, ambulatory. The first week the capacity of the bladder is increased to about 200 cc. by clamping the catheter for one and a half hours. The bladder is then made to contract by scratching the thigh or by some other sensory stimulus. At night the catheter is reattached to the tidal drainage apparatus. The second week increase the time to two hours, then two and a half hours, and finally three hours. The tidal apparatus is then taken off, and the catheter is opened every two hours day and night for a week. The time is increased 30 minutes at weekly intervals to three hours. Removal of the catheter is the next step, and the time between voidings is cut back to two and a

half hours night and day for one week. Then it is increased to every three hours day and night; and after the patient stays dry for a week on this schedule, he may go through the night without voiding. Increases in the day interval may be made up to four hours. Munro had 35 failures out of 125 patients treated with tidal drainage, cystometry, and bladder training.

Manual closed irrigation is simple and foolproof; and if cystometric determinations are carried out to determine the stage of the bladder recovery, it results in bladder control just as tidal drainage does. This method, however, is inaccurate and time-consuming because the bladder capacity may become decreased due to incomplete filling.

The choice between suprapubic or transurethral bladder drainage may be difficult in some cases. We believe that lower urinary tract complications such as urethritis, urethral fistulas, prostatitis, vesiculitis, and epididymitis are avoided by suprapubic cystotomy. These complications may result in urethral stricture and possibly in sterility.

A transurethral catheter may produce fistulas at the peno-scrotal junction if left in for a long time. It may be possible to prevent such fistulas by keeping the penis straight on the abdomen to avoid kinking the urethra at the peno-scrotal junction. The urethra of a paralyzed patient tolerates pressure from an indwelling catheter no better than the skin of the sacrum tolerates body pressure. If the neurologic survey indicates partial transection of the cord, urethral catheter drainage may be tried for a few weeks in the hope that bladder function can rapidly be reestablished. If catheter drainage is required longer than six weeks, it is best to employ suprapubic cystotomy.

To prevent calcium incrustations from forming on the retention catheter, it should be changed at weekly intervals, the urine should be kept acid, a large fluid intake should be encouraged, and infection must be kept at a minimum. Frequent turning of the patient should be insisted upon and may best be accomplished on a Stryker frame. Finally, frequent roentgenograms of the urinary tract must be taken for early detection of stones if they do form.

In patients with urethral stricture or preexisting infection of the lower urinary tract, there is no choice but to do a suprapubic cystotomy. The comprehensive reports of Thomson-Walker and Riches supported by the experiences of Prather during World War II indicate that cystotomy presents no difficulty in the rehabilitation of the bladder to a voiding state. Only occasionally is secondary closure of the suprapubic sinus necessary. Functional results and

bladder capacity are not influenced unfavorably even after months of drainage, provided bladder infection remains minimal.

The suprapubic catheter can be connected to a closed manual irrigation system or to a tidal apparatus, and bladder training as advocated by Munro may be carried out. The patient may be made ambulatory just as with transurethral drainage. When bladder recovery is complete, the suprapubic catheter is removed; and usually the sinus closes promptly. If not, a transurethral catheter may be necessary for a few days.

The technic of cystotomy is important. If properly done, a suprapubic cystotomy does not leak. The bladder is permitted to fill. No anesthesia is necessary in spinal cord injury. A short midline incision is made high over the dome of the bladder through the skin and linea alba. The bladder is stabbed with an F 22 trocar pointed slightly upward to avoid injury to the trigone, and an F 20 Foley catheter with a 5 cc. bag is inserted through the trocar into the bladder. The bag, previously tested for leaks, is inflated, and the trocar is withdrawn. As a precautionary measure, the catheter is fixed to the skin with two black silk sutures which will hold in case the catheter bag breaks. The skin is closed with interrupted black silk. The first catheter change should be made in 10 days. It is then changed every seven days.

Several months after the trauma, delayed treatment for bladder neck obstruction may be necessary. There may be detrusor hypertrophy and hypertrophy of the bladder neck musculature as evidenced by trabeculation of the bladder wall and increasing amounts of residual urine. The bladder neck hypertrophy may be accompanied by sclerosis caused by chronic infection. The bladder is inefficient because it works against an unopened vesical orifice due to the failure of the trigone muscle to function properly. The hypertrophy and/or sclerosis of the bladder neck must be resected regardless of how minimal it may appear. Patients then have a better stream, little or no residual urine, and improved vesical control. It is important to allow a prolonged period for recovery and to do multiple resections if necessary.

When the bladder neck obstruction is relieved and a small quantity of residual urine remains, parasympathetic stimulants are of some service. Drugs used are Pilocarpine, Mecholyl, Urecholine, and Furmethide. The best and most widely used of these at present is Urecholine. It can be given by mouth in doses of 10 to 20 mg. every six hours and has been used as long as three months without harmful effect.

Conclusions

Management of the urinary bladder in the paraplegic is discussed including immediate and delayed treatment. Tidal drainage is helpful but not essential.

Suprapubic cystotomy minimizes many complications.

Transurethral resection of the bladder neck improves bladder function in cases showing bladder neck hypertrophy and/or sclerosis.

Parasympathetic stimulants are helpful to reduce the residual urine in the neurogenic bladder previously relieved of its vesical neck obstruction.

References

1. Cone, W. V., and Bridgers, W. H.: Combined tidal irrigator and cystometer for management of paralyzed bladder, Surg., Gynec. & Obst. *75:*61 (July) 1942.
2. Emmett, J. L.: Transurethral resection in treatment of true and pseudo cord bladder, J. Urol. *53:*545 (April) 1945.
3. Emmett, J. L., and Dunn, J.: Transurethral resection in surgical management of cord bladder, Surg., Gynec. & Obst. *83:*597 (Nov.) 1946.
4. Hanlon, R. E.: Treatment of urinary incontinence in vesical paralysis, J. Urol. *53:*572 (April) 1945.
5. Hinman, F.: Care of bladder at the front when paralyzed by injuries to spinal cord, J. Urol. *46:*499 (Sept.) 1941.
6. Lewis, L. G.: Treatment of bladder dysfunction after neurologic trauma, J. Urol. *54:*284 (Sept.) 1945.
7. Mullenix, R. B.: Cystometry in study of traumatic neurogenic bladder, J. Urol. *55:*470 (May) 1946.
8. Munro, D.: Rehabilitation of patients totally paralyzed below waist, with special reference to making them ambulatory and capable of earning their own living. II. Control of urination, New England J. Med. *234:*207 (Feb. 14) 1946.
9. Munro, D.: Rehabilitation of patients totally paralyzed below waist, with special reference to making them ambulatory and capable of earning their own living. III. Tidal drainage, cystometry and bladder training, New England J. Med. *236:*223 (Feb. 13) 1947.
10. Prather, G. C.: Spinal cord injuries; care of bladder, J. Urol. *57:*15 (Jan.) 1947.
11. Prather, G. C.: The Urological Aspects of Spinal Cord Injuries, Springfield, Ill., Charles C Thomas, 1949.
12. Van Duzen, R. E.: Operations for urinary control of neurogenic bladder, J. Urol. *53:*565 (April) 1945.

FAILURE OF REPERITONEALIZATION TO PREVENT ABDOMINAL ADHESIONS IN THE DOG

ALBERT O. SINGLETON, JR., M.D.
ED. B. ROWE, M.D.
ROBERT M. MOORE, M.D.
Galveston

INTESTINAL obstruction from postoperative adhesions has long been a major problem. Equivocal success has resulted from placing such agents as amniotic fluid and anticoagulants in the peritoneal cavity in the hope of reducing adhesions. More recently cortisone and corticotrophic hormones have been recommended to prevent formation of adhesions. A regime of early motility of the gastrointestinal tract has also been recommended.[2,5,6,7]

It has been recognized for many years that excessive handling of tissues, *i.e.*, trauma, increases the incidence of adhesions. There has also arisen a general belief that reperitonealization of denuded areas after surgery lessens adhesion formation. We have attempted to test this last impression in a series of experiments on the dog.

1. *Peritoneal patches.* Multiple squares of peritoneum, each 1 cm. by 1 cm., were removed from the terminal ileum in 10 animals. In some areas muscularis was removed also. In each animal the more distal areas were covered with patches of peritoneum removed from the abdominal wall or from the mesentery, and fastened in place over the denuded intestinal wall with interrupted No. 0000 silk sutures. In the same animals the more proximal areas were left uncovered. On examination after intervals of two weeks to two months more adhesions were found on the patched areas than on the uncovered control areas. For the most part the control areas were free of adhesions.

2. *Closure of raw areas by simple suture.* In 10 other animals similar areas were denuded of peritoneum. In each animal the more proximal areas were left open but the serosa was closed over the more distal areas using No. 0000 silk, No. 80 cotton, No. 34 wire or No. 000 catgut as suture material. Examination two weeks to four months later revealed more adhesions had occurred on the sutured areas than on the uncovered control areas. In many instances it was difficult to discern that the bowel had been injured at all in the areas where no attempt had been made to peritonealize the defect.

3. *Closure of raw areas by plication.* In five animals denuded surfaces of bowel were covered over by plication. Continuous and interrupted sutures of No. 34 wire, No. 80 cotton, No. 0000 silk, or No. 000 catgut were used for the plication, starting at the mesenteric root and continuing onto the bowel so that the raw areas were covered by folding the intestine upon itself. On sacrificing these animals after intervals of from two weeks to four months, adhesions were found along the suture lines of the plications in greater number than on the untreated control areas in the same animals.

4. *Removal of parietal peritoneum.* In 10 animals large areas of parietal

From the Department of Surgery, University of Texas Medical Branch, Galveston.

peritoneum were excised. No adhesions formed at these areas except in one instance, in which the adhesions were few in number and light in texture. However, adhesions to the suture line where the abdominal wound was closed were seen in almost all of these animals.

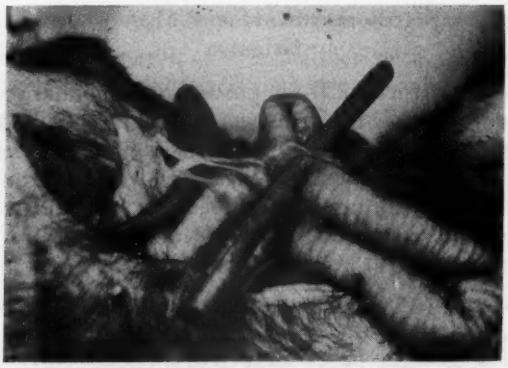


Fig. 1. Adhesions occurring on areas of bowel which were denuded of peritoneum and then closed with No. 0000 interrupted silk sutures. Animal sacrificed 33 days after operation. Rubber tubes are placed in resulting openings through which other loops of bowel could herniate. There were no adhesions to control area where denuded bowel was left untreated.

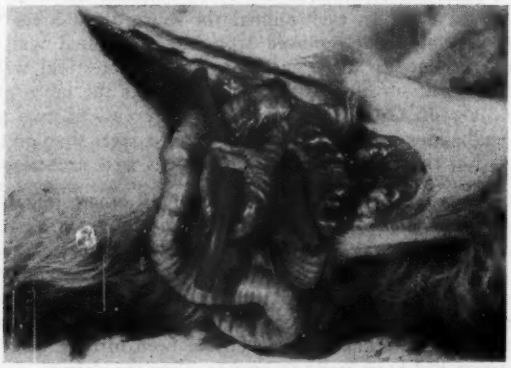


Fig. 2. Adhesions occurring on areas of bowel which were denuded and then peritonealized by approximating serosa with No. 000 interrupted chromic catgut sutures. Adhesions were also growing to control area in this dog. Animal sacrificed after 27 days.

5. *Passage of sutures through peritoneum.* In five animals sutures of No. 0000 silk were passed through denuded areas but were not tied. More adhesions were found later in these areas containing foreign body than in untreated control areas.

6. *Minimal manipulation.* It was noted in all experiments that adhesions were frequent in areas of intestine and mesentery which supposedly had not been traumatized.

Discussion. Chester, Bell and McCorkle,[1] in 1949, covered intestinal anastomoses in the dog with peritoneal grafts in an attempt to obtain additional security at the suture line. The raw surface of

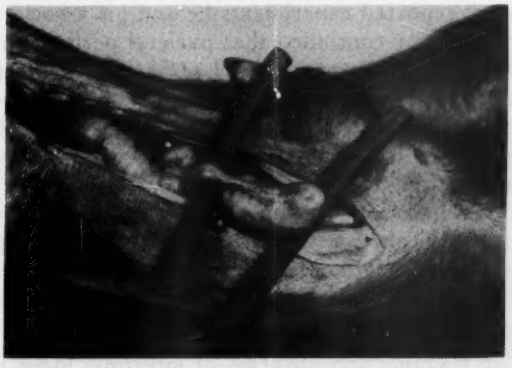


Fig. 3. Adhesions occurring along suture line of plicated loop of bowel. One fine adhesion occurring on denuded control area. Plication with No. 0000 silk continuous suture had been used to cover deperitonealized area on bowel. Animal sacrificed after 27 days.

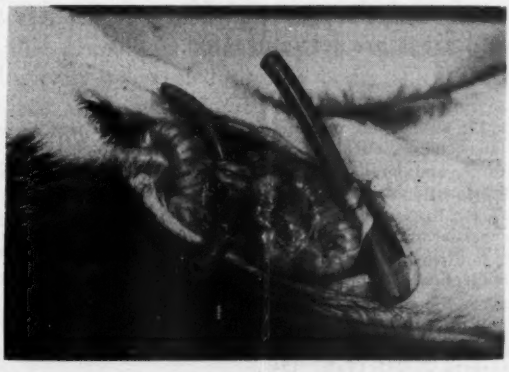


Fig. 4. Adhesions occurring on loops of bowel plicated with No. 000 chromic catgut. Plication was used to cover raw areas of bowel formed after freeing old adhesions. Animal had been operated on three times previously. Sacrificed after 33 days.

the peritoneal graft was placed outward. Adhesions to the grafts were formed in 90 per cent of the animals examined during the first two weeks and in 40 per cent of those examined after six weeks. We found adhesions to be equally numerous when the smooth surface of the peritoneal graft was placed outward.

In 1949 Robbins, Brunschwig and Foote[3] reported experiments in dogs in which areas of parietal peritoneum were removed with a

minimum of adhesion formation resulting. They also reported a series of abdominal operations in human beings in which large areas were left without peritoneal covering; postoperative intestinal obstruction from adhesions did not result. In discussing these experiments Rhodes[4] reported experiments he and his associates had conducted in rats. They concluded that parietal peritoneum would regenerate in the rat, as would also visceral peritoneum provided it was removed with great care to avoid injury to the underlying layers. They also found that when fine silk sutures were placed under the peritoneum and left untied no adhesions resulted, whereas oversewing denuded areas on the rat's cecum with the same suture material often resulted in formation of adhesions. Denuded areas left untreated formed adhesions less frequently. Rhodes suggested that reperitonealization would occur spontaneously in man with less formation of adhesions than when raw areas were sutured.

Peritonealization of gastrointestinal suture lines may be of value in preventing leakage, and plication of intestinal loops may be useful in preventing obstructing angulations. We have been unable to find evidence in the dog, however, that any form of reperitonealization lessens the incidence of adhesion formation. On the contrary, the results in this study suggest that adhesions are less apt to form when denuded areas are left untreated.

REFERENCES

1. Chester, S. T.; Bell, H. G., and McCorkle, H. J.: Use of free peritoneal grafts in intestinal anastomoses, Surg., Gynec. & Obst. *89:*605 (Nov.) 1949.
2. Lehman, E. P., and Boys, F.: Experimental prevention of intraperitoneal adhesions with heparin; third report, Surgery *12:*236 (Aug.) 1942.
3. Robbins, G. F.; Brunschwig, A., and Foote, F. W., Jr.: Deperitonealization: clinical and experimental observations, Ann. Surg. *130:*466 (Sept.) 1949.
4. Rhodes, J., in discussion on Robbins, G. F.; Brunschwig, A., and Foote, F. W., Jr.: Deperitonealization, Ann. Surg. *130:*478 (Sept.) 1949.
5. Scheinberg, S. R., and Saltzstein, H. C.: Effect of cortisone and corticotrophin (ACTH) on intra-abdominal adhesions, Arch. Surg. *63:*413 (Oct.) 1951.
6. Schiff, C. A.; Goldberg, S. L., and Necheles, H.: Prevention of abdominal adhesions, Surgery *25:*257 (Feb.) 1949.
7. White, B. H.: Effect of dicumarol upon postoperative peritoneal adhesions, Ann. Surg. *130:*942 (Nov.) 1949.

EOSINOPHILIC GRANULOMA OF THE SKULL

Report of Six Cases

GUSTAVUS A. BYNUM, M.D.
Galveston

THIS paper deals with a peculiar bone lesion which has been recognized as a definite clinical entity for only about 12 years. Lesions of a smilar type had been described by Finzi in 1939, Mignon in 1930 and Shairer in 1938. They did not consider the disease to be a distinct entity, referring to it as "myeloma with prevalence of eosinophilic cells," "granulation tumor of bone," and "osteomyelitis with eosinophilic reaction" respectively. General recognition was not attracted to the lesion, however, until 1940 when Lichenstein and Jaffe first clearly defined the condition as a clinical entity and suggested the use of the descriptive name "eosinophilic granuloma."[8,12] In 1944 Lichtenstein and Jaffe reevaluated the microscopic features of the disease and concluded that Letterer-Siwe's disease, Schüller-Christian's disease, and "eosinophilic granuloma" may be considered as different clinical and anatomical expressions of the same basic disorder.[8]

Eosinophilic granuloma appears to be clinically, histologically and pathologically a benign lesion of bone which most commonly affects children and young adults, about 64 per cent of cases occurring in patients less than 20 years of age.[4] The exact etiology remains unsettled. Otani and Erhlich suggested trauma as a causative factor.[15] Lichenstein and Jaffe believe the disease may be due to a virus, with the portal of entry in the intestinal tract.[8] Most authors agree that it is of inflammatory origin but no definite organism has been demonstrated.[13]

The literature reports about an equal sex incidence. The disease is more common in childhood but is not limited to children and young adults, cases being reported in patients 6 months to 58 years of age.

Almost without exception eosinophilic granuloma is a disease of bone, though there are recent reports in the literature of extraosseous lesions.[1,3,4,6,20] Bone lesions are usually solitary and may involve the bones of the skull, pelvis, long bones, vertebrae and ribs. Multiple lesions are not uncommon, and 1 case is reported in which there were 25 separate lesions. In a review of 53 cases the lesions were solitary in 36 and multiple in 10 cases. The skull was

From the Department of Surgery, Division of Neurosurgery, University of Texas Medical Branch, Galveston.

the site of single lesions in 36 per cent of cases and multiple lesions in 11 per cent.[4,16,20]

Both local and systemic signs and symptoms of slight to moderate degree are present in most cases. The most common local symptoms are pain, swelling of the overlying soft tissues, and tenderness at the site of the lesion. Among local complaints arising from the skeletal lesions, local tenderness without pain is the most common. A lesion in the femoral neck will be associated with a limp and one of the vertebral column with a deformity of the spine. When multiple lesions are present, only one may produce symptoms.[6,8,20]

Systemic manifestations such as slight fever, general malaise, and weight loss are sometimes present. There may be a leukocytosis with eosinophilia. With these findings a correct diagnosis may be reached. It is more common, however, for the disease to be relatively silent until roentgenologic examination reveals a destructive bone lesion. This finding usually follows a history of obscure pain and weight loss such as might be associated with a malignancy.[8,13]

The laboratory examination usually shows a leukocytosis with an eosinophilia of variable degree. The eosinophilic count in some reported cases did not return to normal for one and one-half to three years after removal of the lesion. Likewise sternal marrow punctures have shown an increase in eosinophilic myelocytes for as long as three years after curettement of the lesion. Chemical determinations for blood calcium, phosphorus, cholesterol, cholesterol esters and total lipids have consistently yielded normal findings. Tissue studies for bacteria and viruses, cultures, and animal inoculations have all been negative.[4,6,8]

The individual bone lesion presents itself as a large or small area of radiolucence. In the calvarium it appears as a sharply demarcated area of rarefaction with a punched-out appearance. Extensive resorption of both tables, particularly the outer, is associated with a swelling of the overlying soft tissues. The lesion apparently originates in the medullary cavity and as it grows the cortex may be rarefied, perforated, and occasionally expanded. Pathologic fractures are seen in the ribs, vertebrae and long bones.[4,6] Generally there is nothing distinctive about the individual lesions. For this reason the solitary bone lesion, because of rapid onset and absence of systemic manifestations, may suggest a primary malignant tumor. With multiple lesions the roentgenogram may suggest metastatic malignancy, multiple myeloma, or Ewing's tumor.[4,6,8]

The gross appearance of the lesion in the early stage is that of

a soft, friable, hemorrhagic and cystic, yellowish-brown and red material. The yellow color is due to necrosis and the red to hemorrhage. In the later stages the cysts are replaced by friable yellow tissue which is softer than the surrounding bone. As healing proceeds there is replacement by gray connective tissue and ultimately by bone formation. In skull lesions the granulation tissue will often be found attached to the dura on the inside and to the pericranium on the outside of the points of bone perforation.[8,14] Baker and Fisher reported a case (1948) with direct invasion of the cortex of the brain.[1]

Histologic examination shows a destructive granulomatous lesion characterized by large sheets of phagocytic histiocytes intermixed with numerous eosinophilic leukocytes and reactive multinucleated giant cells. The phagocytic cells are especially abundant in areas of necrosis and hemorrhage. The histiocytes are large uniform cells with oval, pale-staining nuclei. Erythrocytes, eosinophilic granules, and hemosiderin pigment may be found in the cytoplasm of the histiocytes and giant cells. The eosinophiles, though not the basic constituent of the lesion, are responsible for the striking picture of the disease. In the later stages the eosinophiles tend to disappear leaving a predominance of mononuclear cells and fibrosis. Neutrophilic leukocytes, plasma cells, and lymphocytes may be found in small numbers.

The diagnosis cannot be made except by histologic examination of tissue removed from the lesion. Frozen biopsy section is not always specific, and, since the lesion resembles a neoplasm, wide surgical excision has been the treatment of choice in solitary lesions. Single lesions in the skull and long bones are best treated by thorough curettement. A more radical procedure than local excision is not indicated. Solitary lesions treated by curettement alone show gradual repair of the affected area over a period of several months. In most cases supplementary treatment with irradiation has been given, but it is not certain that roentgenotherapy hastens the healing process as 2 cases are reported in which new lesions developed after therapy had been initiated.[6,20] When multiple lesions are present irradiation has been largely the treatment of choice once the diagnosis has been made by curettement and biopsy specimen.

Eosinophilic granuloma of bone offers an excellent prognosis in spite of its rapid development and destructive nature. Postoperative follow-up examinations over periods ranging from seven months to nine years have shown complete relief of symptoms and gradual bone repair. The postoperative reports and clinical observations on cases treated by curettement, by excision, and by irra-

diation have all been encouraging regardless of the method of treatment. Cases with multiple bone lesions offer a more guarded prognosis since they may be associated with the visceral lesions seen in Letterer-Siwe's disease or in Schüller-Christian's Disease.[4,6,7]

The former is usually a fatal disease of infants and young children manifested by a rapid febrile course, lymphadenopathy, hepatomegaly, splenomegaly, purpura and severe anemia.[17]

Case Reports

The basis of this report is a series of 6 cases believed to answer the criteria established for the diagnosis of eosinophilic granuloma of bone. The patients were treated on the Neurosurgical Service of the John Sealy Hospital during the 12-year period between April 1940 and February 1952. With the exception of 1 case all were solitary lesions. There were three males and three females, the age varying from 18 months to 49 years. One case has been followed for nine years and another for five years; both are well and in good health and have shown no evidence of recurrence.

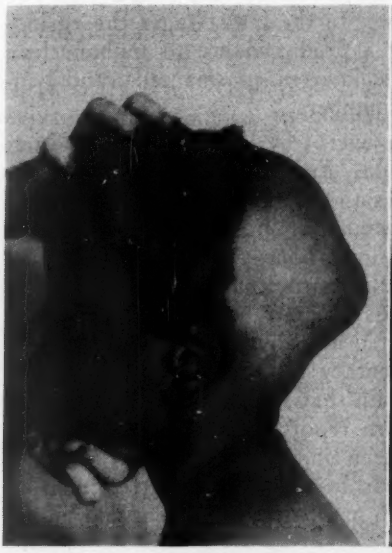


Fig. 1. This photograph shows the size and location of the lesion described in case 1 of this series.

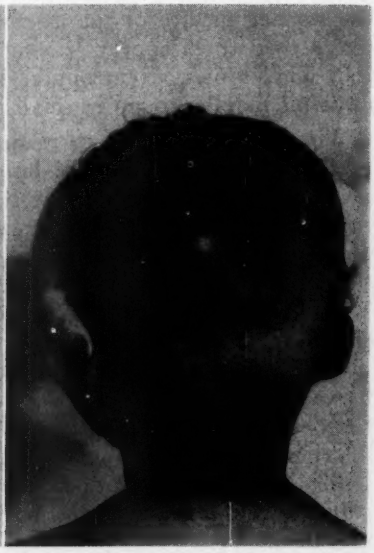


Fig. 2. This photograph illustrates the appearance of the mass on the posterior aspect of the head prior to surgery, case 1.

Case 1. A male Negro child of 18 months was admitted to the hospital on April 29, 1940, with the chief complaint of a "lump" on his head of six months' duration. Four months before admission, the lump began to increase rapidly

in size. On physical examination the child did not appear ill and his temperature was normal. Positive findings were limited to the head which showed a rounded, hard mass in the posterior parietal region just to the left of the midline. This non-tender swelling measured 7 cm. in diameter and 2 cm. in thickness and showed no signs of inflammation. The neurologic examination was normal.

The blood calcium was 11 mg. per cent, the phosphorus 6.54 mg. per cent, and the cholesterol 181 mg. per cent. Repeated blood counts showed a leukocytosis of 9,000 to 11,000 per cu. mm. with an eosinophilia ranging from 18 per cent to 52 per cent. A roentgenologic and bone survey showed only an eroded defect somewhat circular in outline measuring 25 by 8 mm. in the left parietal bone near the junction of the saggital and lambdoid structures.

On May 17, 1940 a bone flap was turned down over the posterior parietal region to include a tumor mass with poorly defined edges. This tumor mass was attached to the pericranium and was largely superficial to the skull. Two small holes in the bone measuring 10 and 5 mm. in size and oval in shape were noted about the center of the tumor mass. The tumor itself measured 5 cm. in diameter and was separated from the bone flap by sharp dissection. It cut with great resistance except at the points of bone perforation where the tumor appeared necrotic and degenerated and contained a fluid material strongly suggesting an inflammatory origin. The tumor was attached to the dura at the points of bone perforation. This area of the dura was electro-coagulated. The bone flap itself was boiled and replaced and the wound closed without drainage. The routine biopsy sections were reported as subacute and chronic inflammation, type not specific.

The biopsy sections on this case have been reviewed and conform to the histologic picture of eosinophilic granuloma. The patient was discharged on June 7, 1940, with the wound completely healed. No roentgenotherapy was given as the true nature of this lesion was not then appreciated. Subsequent visits have shown no spread or recurrence of the process. A recent conversation with the mother of this patient reveals that he has had no further difficulty with reference to the lesion. He is now 12 years of age and in the fifth grade in school.

CASE 2. A 13 year old girl was admitted to the hospital on Oct. 18, 1944 with the chief complaint of a draining sinus on the back of her head. She had been in good health until 15 months before admission when she noticed a non-tender lump on the back of her head while combing her hair. Within a month this mass had reached the size of a hen's egg and was reddish purple in color. Three months after its onset the mass was aspirated by the family doctor and a bloody, non-purulent material obtained, which was negative on smear and culture. After aspiration the lesion had continued to drain a small amount of material which occasionally contained fragments of bone.

Physical examination revealed a draining sinus at the midline of the skull near the junction of the saggital and lamboid sutures. There was a considerable amount of what appeared to be granulation tissue, together with a bloody purulent discharge in the center of the area. The scalp was seen to pulsate vigorously in the region indicating that the bone had been destroyed. Neurologic examination was normal.

Laboratory studies showed normal values for red blood cells and hemoglobin. The leukocyte count was 12,000 per cu. mm. with a normal differential. The blood calicum was 9.8 mg. per cent, phosphorus 4.69 mg. per cent, and

cholesterol 200 mg. per cent. A smear from the draining sinus culture showed staphylococci, but this was considered a contaminant. Roentgenograms of

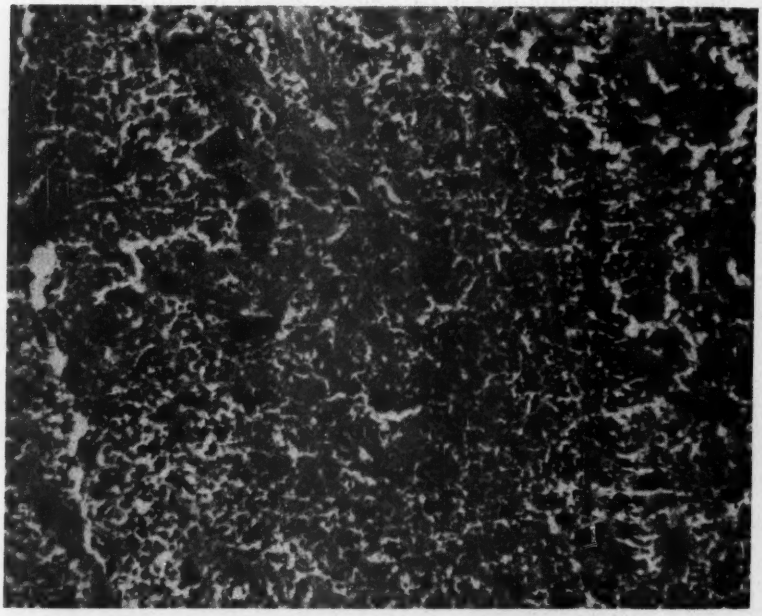


Fig. 3. Low power photomicrograph of the lesion in case 3 showing typical area of granulomatous inflammation with multinucleated giant cells, red blood cells, histiocytes and a large number of eosinophiles. (150X).

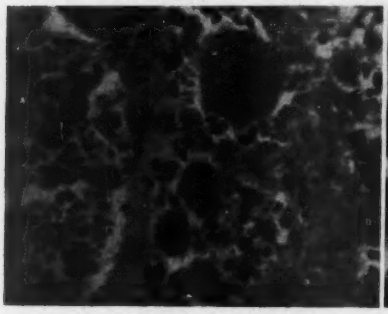


Fig. 4. High power photomicrograph showing typical multinucleated giant cells, large histiocytes and many darkly stained eosinophiles, case 2 (645X).

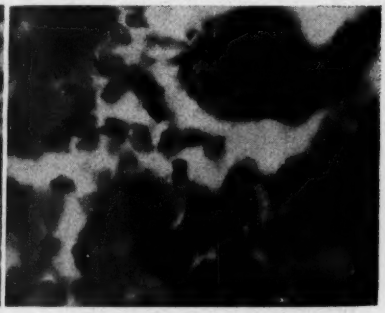


Fig. 5. High power photomicrograph showing hemosiderin granules and cellular debri in the cytoplasm of the histiocytes. The latter are large pale staining cells with oval or rounded single or multiple nuclei, case 2 (1425X).

the skull showed an area of bone destruction roughly circular in outline and measuring 8 cm. in diameter involving both occipital and parietal bones. The

edges were moth-eaten and showed no periosteal bone reaction. A bone survey was normal.

On Nov. 9, 1944, the lesion was explored. The mass appeared brownish red in color and was thought by the operator to be tumor rather than osteomyelitis of the skull. The tumor was firmly adherent to the dura. A frozen biopsy section was reported as showing no evidence of a neoplasm and excision of the involved dura was not done. The dura was curetted and the entire mass removed.

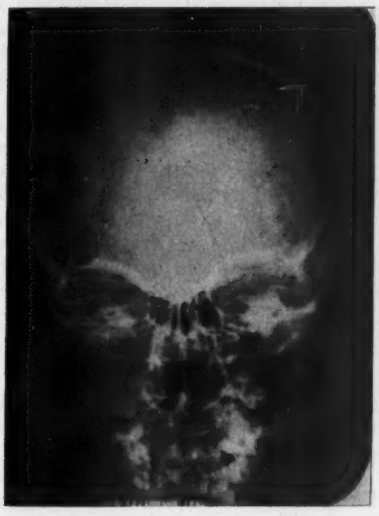


Fig. 6. Roentenogram of the skull showing large defect in occipital and parietal bones which measured 8 cm. in diameter, case 2.

The patient did well postoperatively and the wound healed throughout except in the area of the old sinus tract from which a small amount of drainage continued. The histologic examination showed findings typical of eosinophilic granuloma. The patient was given a total of 2000 u. roentgen rays postoperatively. The drainage from the old sinus tract had diminished, but healing was not complete at the time of her discharge.

A subsequent visit seven months after leaving the hospital showed complete healing of the skin and the skull defect appeared to have closed completely as no pulsation was noted and no defect could be palpated. There was no roentgenologic evidence of recurrence.

CASE 3. A white boy of 7 years was admitted to John Sealy Hospital on Dec. 15, 1948, complaining of a lump on his head, of two and one-half months' duration. There was no history of pain or tenderness. The mass had gradually increased in size until at the time of admission it was about the size of a lemon. Bloody fluid had been aspirated by the referring physician, and roentgenogram of the skull which was made by him revealed a defect in the skull underlying the mass. The patient did not appear acutely ill. Examination of the head revealed a soft, fluctuant, non-tender swelling in the left

parietal region measuring 3 by 4 cm. and elevated above the surrounding scalp for ½ to ¾ cm. Roentgenograms of the skull showed a defect in the left parietal bone which extended through both tables. A bone survey failed to show any other lesions.

The laboratory examination showed 6,600 leukocytes per cu. mm. with polymorphonuclear neutrophiles, 41 per cent, lymphocytes, 46 per cent, monocytes 2 per cent, and eosinophiles, 6 per cent. Urine examination for Bence-Jones protein was negative.

The patient was operated upon on Dec. 16, 1948, and a well encapsulated tumor mass was exposed. This mass was found to lie beneath the pericranium and was easily separated from it. The tumor had a smooth surface, a reddish brown color, and was soft in consistency. It was found to spread out upon the outer surface of the bone in all directions for a distance of 1 cm. or more about the bony defect. It was easily removed from the external surface of the skull as well as from the defect. There was also a tendency for the tumor to spread out from the defect on the internal surface of the bone. The defect in the skull was enlarged until normal-appearing dura was seen. The tumor tissue was attached to the dura, and was curetted away with some difficulty. Excision of this portion of the dura seemed desirable, but a frozen biopsy section was reported to be granuloma. The wound was closed without drainage. Routine biopsy sections revealed an eosinophilic granuloma.

The postoperative course was uneventful. He was discharged on the second postoperative day to return for roentgenotherapy. Subsequent visits showed satisfactory wound healing and no evidence of recurrence.

CASE 4. A white woman, aged 33 years, was admitted to the hospital on Feb. 28, 1949, with the chief complaint of a "cyst" on her head. Three months before admission she had noted a tender area over the frontal scalp when she combed her hair. One month later she noted the presence of a soft tender mass in the frontal region. This continued to enlarge and wearing a hat became painful. Her physician prescribed warm wet compresses and sulfa drugs without benefit. The lesion was then incised and a bloody purulent material obtained. The scalp healed, but the lesion recurred and was reopened three or four times during the two months before her admission. She was referred to another physician who advised her, after biopsy specimen, that she had a bone cyst.

Physical examination revealed a mass, 4 by 4 by 1.5 cm., soft, fluctuant, somewhat tender, over the left frontal bone anterior to the coronal suture. There was absence of hair and the presence of a healed scar over the center of this mass. There were a few tender enlarged lymph nodes in both anterior cervical regions. The temperature was normal. Roentgenograms of the skull showed a circular punched-out area of bone destruction in the left frontal bone involving both inner and outer tables.

The laboratory examination showed the urine to be normal. The white blood count showed a total of 8,550 cells per cu. mm. with a normal differential and no eosinophiles. There was a slight anemia with 3.85 million erythrocytes and hemoglobin, 78 per cent. The Kahn and Kolmer tests were negative. A presumptive diagnosis of eosinophilic granuloma of bone was made and the scalp mass was explored on March 1, 1949. The main mass of the lesion was found beneath the pericranium. It was yellowish red in color and the central portion was somewhat necrotic consisting of 10 to 15 cc. of yellow material similar to pus. Smear and culture showed no organisms. The bone was

rongeured away until the involved portion about the defect was removed. The tumor tissue was strongly attached to the surface of the dura and was curetted away. The dura was opened at the point of greatest attachment, but no evidence of extension was seen. A frozen biopsy section was reported to be granuloma. The scalp was closed without drainage.

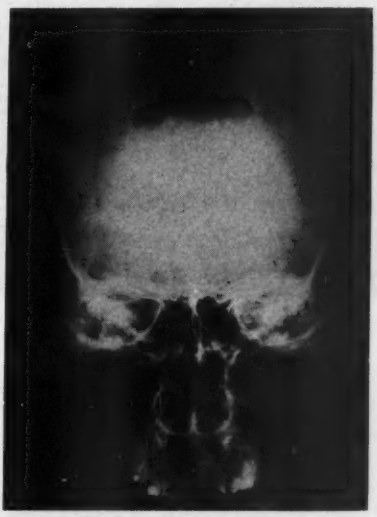


Fig. 7. Roentenogram of the skull showing the eroded "moth-eaten" appearance of the frontal bone produced by the lesion in case 4.

Postoperatively the wound healed well without infection. Small quantities of serosanguineous fluid were aspirated on two occasions. This was thought to be spinal fluid which had leaked through the dural incision. The final pathologic report was eosinophilic granuloma. She was discharged 10 days postoperatively to receive roentgenotherapy. She was readmitted in November 1949 for a tantalum cranioplasty at the frontoparietal defect.

The patient returned on Feb. 4, 1952 with an additional lesion in the posterior parietal region measuring 1.5 cm. in size. Roentgenologic findings were consistent with the diagnosis of granuloma and she received 1800 u roentgen rays as an out-patient with disappearance of the mass, but with little bone regeneration in six weeks. She was discharged from treatment on March 24, and has not been seen for follow-up examination.

CASE 5. A 47 year old white woman was admitted to the hospital on March 2, 1949, with the chief complaint of a tender lump on the right side of her head of three months' duration. The presence of this lump had been associated with intermittent right temporal headaches and a "shooting" pain on touching the mass. Headaches were relieved by mild analgesics such as aspirin. Physical examination revealed a moderately obese, graying, woman who did not appear ill. Positive findings were limited to an area of tenderness over the right temporal bone about the size of a 50 cent piece. The central portion of this area was elevated 3 to 4 mm. above the surrounding scalp by

a small firm mass. Several other small tender areas were present on careful examination of the scalp.

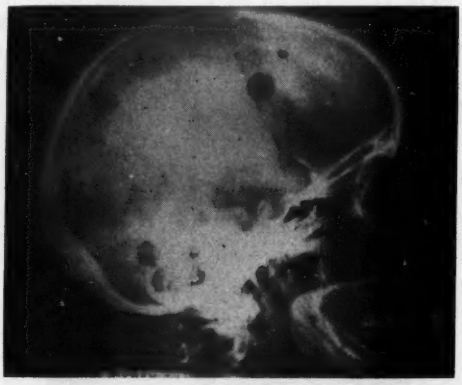


Fig. 8. Roentgenogram of the skull showing multiple areas of bone destruction seen in case 5. Smaller areas on the film are not reproduced by this photograph.

Fig. 9. Low power photomicrograph from an area showing bone trabeculation, areas of necrosis and infiltration with inflammatory cells. The darkly staining cells are eosinophilic leukocytes, case 5 (114X).

The laboratory examination showed 5.3 million erythrocytes per cu. mm. with 103 per cent hemoglobin, and 14,600 leukocytes per cu. mm. with eosinophiles, 5 per cent. The urinalysis was normal. Both Kahn and Kolmer tests were negative.

The roentgenologic examination of the skull revealed multiple small areas of bone destruction in the right temporal bone near the coronal suture. Several additional small rounded areas of bone destruction were noted in the bones of the calvarium suggesting a metastatic malignancy. A bone survey showed no other areas of bone involvement. Both metastatic malignancy and multiple myeloma were considered possibilities.

On March 3, 1949, the patient was prepared for a biopsy specimen. After shaving the head, the mass which had been barely palpable was easily seen in the right temporal area. The lesion was found beneath the pericranium as a flat mass of grayish white tissue measuring 1.25 cm. in diameter and 2 to 3 mm. in thickness. This tissue was scraped from the bone and sent to the pathologist for frozen biopsy section. Close inspection of the underlying bone revealed it to be honeycombed by 10 to 20 small, pin-point openings. This area of bone was removed with a Becker trephine and the inner surface found to be irregular. The underlying dura showed a small area of yellowish gray tissue similar in appearance to that found beneath the pericranium. The tissue was curetted away and sent to the pathologist. The frozen biopsy section was reported as showing no evidence of malignancy but was a granuloma. The final biopsy sections were reported eosinophilic granuloma.

The patient was discharged home on the day following operation to receive roentgenotherapy at home from the referring physician. She did not report for a follow-up examination.

Fig. 10. The sharply localized character of bone destruction in the posterior parietal region is well illustrated in this roentgenogram of the skull in case 6.

CASE 6. An 11 year old white girl was admitted to the hospital on June 20, 1949, with the history of a lump on the back of her head of six weeks' duration. It increased in size from ½ inch to 2 inches in diameter in two weeks. The family physician regarded the lesion as a cyst until roentgeno-

grams of the skull revealed what was interpreted as a "fracture with a blood clot." The lump was incised by the referring physician. A biopsy specimen was reported to be malignant and the patient was referred with the diagnosis of a sarcoma of the scalp.

Physical examination revealed a pulsating, soft, slightly tender mass, 3 cm. in diameter, just to the left of the midline in the occipital region. There was a serous drainage containing soft, yellow necrotic tissue in the center of the area. Several small, firm, discrete lymph nodes were noted in both posterior cervical triangles. Roentgenologic examination revealed a circular punched-out area of bone destruction extending through both tables of the occipital bone. A bone survey showed no similar lesions elsewhere.

Blood counts, urinalysis, serologic tests, and blood-chemical determinations were within normal limits. The neurosurgical consultant was of the opinion that the child had an eosinophilic granuloma and curettement of the lesion was advised.

On June 23, 1949, the lump was explored, and reddish gray tissue was exposed beneath the pericranium. The bony defect was occupied by a tumor-like, yellowish red to brownish red tissue, which had attached itself to the dura and which seemed to spread out peripherally under the skull. The tissue was curetted from the dura and pericranium and the entire mass removed. The bone edges were rongeured away until normal dura was seen. The skin edges were debrided and the wound closed. Postoperatively the patient did well and the wound healed. The tissue removed showed the typical picture of eosinophilic granuloma. The patient was given postoperative roentgenotherapy. Six weeks after discharge a small draining sinus developed and she was readmitted. The sinus closed on conservative treatment. She was doing well on her last clinic visit.

Summary

The disease discussed in this paper is a relatively new entity, having been recognized first and described adequately in 1940. It is a disease of the skeleton of children and young adults. In the majority of cases the disease presents itself as a single, well localized lesion beginning in the medullary cavity, and enlarging to erode, expand, and perforate the involved bone. The bone is replaced by a brownish or yellowish granulation tissue showing hemorrhagic areas. The distinctive histologic feature is the presence of tumor-like aggregates of large phagocytic cells intermixed with numerous eosinophilic leukocytes. Eosinophilic granules, red blood cells, and hemosiderin pigment may be seen in the cytoplasm of the large phagocytic cells. There is also a scattering of reactive multinucleated cells.

The etiology of the disease remains obscure. Some suggest trauma as the etiologic agent, but it is probably a virus granuloma. The eosinophilic myelocytes and granulocytes in the sternal marrow suggests a systemic response to an etiologic agent. This is further suggested by the leukocytosis and eosinophilia in the peripheral blood. The disease usually runs a rapid clinical course, character-

ized by a soft, tender swelling associated with destruction and perforation of the underlying bone. In the skull both tables are destroyed and the lesion tends to spread out peripherally under the pericranium externally and upon the dura internally. In spite of the rapid course and the wide areas of bone destruction seen in this disease, the prognosis seems to be uniformly good. Surgical removal of the lesion with or without roentgenotherapy has been effective in all reported cases.

REFERENCES

1. Baker, G. S., and Fisher, R. G.: Eosinophilic granuloma of skull; report of four cases with five-year follow-up, S. Clin. North America *28:*1057, 1948.
2. Bell, T. A.: Eosinophilic granuloma of clavicle; case report, Treat. Serv. Bull. *6:*200 (April) 1951.
3. Brunner, H.: Eosinophilic granuloma of mouth, pharynx, and nasal passages, Oral Surg. *4:*623 (May) 1951.
4. Dundon, C. C.; Williams, H. A., and Laipply, T. C.: Eosinophilic granuloma of bone, Radiology *47:*433 (Nov.) 1946.
5. Farinacci, C. J.; Jeffrey, H. C., and Lackey, R. W.: Eosinophilic granuloma of lung, U. S. Armed Forces M. J. *2:*1085 (July) 1951.
6. Hamilton, J. B.; Barner, J. L.; Kennedy, P. C., and McCort, J. J.: Osseus manifestations of eosinophilic granuloma; report of nine cases, Radiology *47:*445 (Nov.) 1946.
7. Henderson, E. D.; Dahlin, D. C., and Bickel, W. H.: Eosinophilic granuloma of bone, Proc. Staff Meet., Mayo Clin. *25:*534 (Sept. 13) 1950.
8. Jaffe, H. L., and Lichtenstein, L.: Eosinophilic granuloma of bone, condition affecting one, several, or many bones, but apparently limited to skeleton and representing mildest clinical expression of peculiar histiocytosis also underlying Letterer-Siwe's disease and Schüller-Christian disease, Arch. Path. *37:*99 (Feb.) 1944.
9. Kipp, H. A., and Fisher, E. R.: Eosinophilic granuloma of rib; report of case, J. Thoracic Surg. *21:*24 (Jan.) 1951.
10. Kruger, G. O., Jr.; Prickman, L. E., and Pugh, D. G.: So-called eosinophilic granuloma of ribs and jaws associated with visceral (pulmonary) involvement characteristic of xanthomatosis, Oral Surg. *2:*770 (June) 1949.
11. Lapidus, P. W.; Slobody, L. B.: Germansky, G., and Willner, M. M.: Eosinophilic granuloma of bone; report of three cases, Am. J. Dis. Child. *75:*900 (June) 1948.
12. Lichtenstein, L., and Jaffe, H. L.: Eosinophilic granuloma of bone, with report of case, Am. J. Path. *16:*595 (Sept.) 1940.
13. Maurer, E., and DeStefano, G. A.: Eosinophilic granuloma of rib, J. Thoracic Surg. *17:*350 (June) 1948.
14. Meredith, J. M., and Price, W. T., Jr.: Eosinophilic granuloma of skull; report of four cases of benign bone lesion, Virginia M. Monthly *79:*39 (Jan.) 1952.
15. Otani, S., and Ehrlich, J. C.: Solitary granuloma of bone simulating primary neoplasm, Am. J. Path. *16:*479 (July) 1940.
16. Parkinson, T.: Eosinophilic xanthomatous granuloma with honeycomb lungs, Brit. M. J. *1:*1029 (June 11) 1949.
17. Schafer, E. L.: Nonlipid reticulo-endotheliosis; Letterer-Siwe's disease; report of three cases, Am. J. Path. *25:*49 (Jan.) 1949.
18. Sleeper, E. L.: Eosinophilic granuloma of bone; its relationship to Hand-Schüller-Christian and Letterer-Siwe's diseases with emphasis upon oral symptoms and findings, Oral Surg. *4:*896 (July) 1951.
19. Walker, D. G., and Lucas, R. B.: Eosinophilic granuloma, Proc. Roy. Soc. Med. *43:*717, 1950.
20. Weinstein, A.; Francis, H. C., and Sprofkin, B. F.: Eosinophilic granuloma of bone; report of case with multiple lesions of bone and pulmonary infiltration, Arch. Int. Med. *79:*176 (Feb.) 1947.

RESTORATION OF THE SKIN OF THE MALE EXTERNAL GENITALIA FOLLOWING TRAUMA

CLIFFORD C. SNYDER, M.D.
Galveston

IN TRACING the history of the problem of external genitalia skin loss, it is interesting to observe the trend of change in etiologic factors and to note that an increased incidence in this type of trauma since the report by Gibbs[22] in 1855 has followed the development of mechanization in industry and agriculture.[15,31,33,34,38,40,52,53] A report by Judd[28] in 1943 showed that two-thirds of the cases of external genitalia avulsion wounds seen at the Mayo Clinic were due to farm machinery, and Bruner[9] states that, "The substitution of mechanical horsepower for old-fashioned 'power-of-the-horse' on American farms is responsible for many disastrous accidents." Chief offenders among modern farm implements are tractors,[11] harvesters,[17] corn huskers and milling machines.[44,55] Other types of power-driven apparatus with unprotected rotary drive shafts such as washing machines, printing presses, sawmills, ice cream machines, and motor launches have taken their toll.[14,15,32,41,43] Burns likewise are frequently responsible for external genitalia skin loss. These burns vary in source from hot liquids to shortcircuits in high frequency currents.[3] One must not disregard the injudicious use of drugs, and this is appropriately exemplified in the unfortunate case of the devoted housewife who, while attempting to rid her husband of pediculosis pubis, utilized lysol packs with resultant loss of the scrotal and pubic skin. Other unusual accidents are mentioned in the literature. Among these is the case of a sailor who, during a friendly scuffle aboard ship, became aware of an avulsion of his penile skin only because he noticed his blood-stained trousers and not because of pain.[50] A 5 year old boy reported by Robinson *et al.*[42] tied one end of a string around his penis and the other end to the bedpost; when he fell out of bed, the skin of the penis was avulsed. Certain diseases[23] may also be factors in the loss of genitalia integument, as for example: lymphogranuloma venereum,[7,16,37] nonspecific urethritis,[25,37] erysipelas,[19,24,32] elephantiasis,[16] and cancer.[35] Finally, a group of cases following operations for inguinal hernia[55] and redundant prepuce (circumcision) has been reported.[2,8,12,26,32,45,48] In 1 case[42] an elastic band utilized as a tourniquet around the penile base produced a total slough.

The skin covering the penis has a characteristic thinness, absence of adipose tissue and absence or rarity of hair. Injuries to this

From the Department of Surgery, Plastic and Maxillofacial Division, University of Texas Medical Branch, Galveston.

organ are usually superficial and limited to the integument since it is freely movable and poorly attached to Colles' fascia, the subjacent structure. Only at the corona of the glans is the skin firmly adherent and with an avulsion injury a remnant usually remains attached in this area.

The scrotal skin differs in character from that of the penile shaft. Although it is very thin, it is provided with sebaceous glands and hairs, composed of many folds or rugae, and is firmly adherent to the dartos tunic of muscle fibers which glide upon a layer of delicate areolar tissue. Beneath this lies the intercrural fascia, cremaster muscle, infundibuliform fascia and tunica vaginalis. In trauma to the scrotum the skin and dartos are usually removed together because of their firm attachment to each other.

It is interesting to observe that the initial reaction in traumatic denudation of the genitalia is as much a psychologic as a physiologic phenomenon. As a matter of fact, the patient when seen immediately following the accident usually exhibits few if any signs or symptoms of shock, unless he has lost a great deal of blood or has received severe associated trauma. With avulsion of the skin of the penis or the entire organ the patient usually is concerned about his potentia coeundi and appearance whereas, the patient who experiences a scrotal avulsion is concerned with his potentia generandi. Although Owens[37] mentions that older men with families are unconcerned and may welcome infertility. Baxter,[1] in a well organized treatise on this subject, notes that the degree of mental trauma is probably inversely proportional to the patient's age. Young male adults present deep concern over their loss and therefore require immediate and careful psychiatric evaluation and therapy. If the patient has lost only one testis then he may be reassured with regard to hormonal activity and spermatogenesis. But with the misfortune of losing both testes, endocrine therapy may be required. If the gonads are removed from the body before puberty, hormonal substitution treatment should be introduced as early as possible to prevent loss of secondary sex characteristics and additional hormonal upsets such as a tendency to gigantism because the bony epiphyses are late in uniting.

The operative approach to resurfacing the external genitalia is the same as in any other surgical problem: to replace the loss as nearly as possible to the normal in a simplified surgical manner without jeopardizing the result. The surgical technics *per se* are as varied as there are articles on the subject; almost all of them have been reported satisfactory so far as postoperative results are concerned.

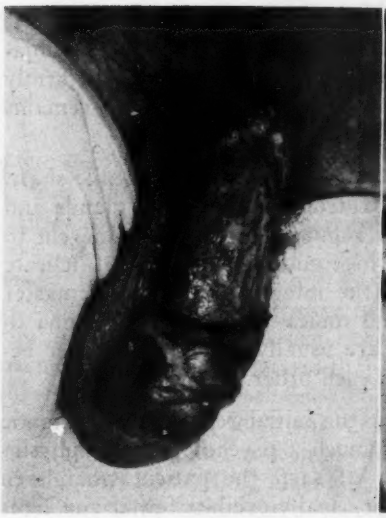


Fig. 1a. S. C. H. 46 year old white oil field worker caught clothes and penis in rotating oil well shaft with resultant loss of entire penile skin with exception of preputial collar.

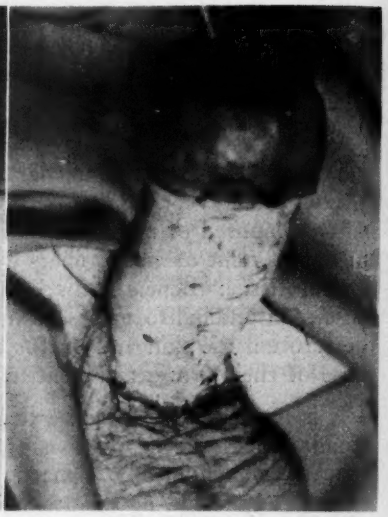


Fig. 1b. S. C. H. Four days postoperative. Illustrating zig-zag line of closure on ventral surface and tie over sutures that have been cut in removing bandage.

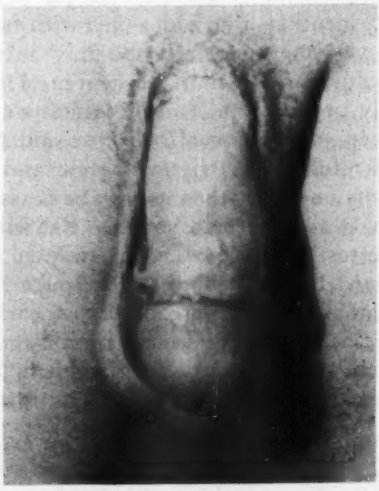


Fig. 1c. S. C. H. Eleven days postoperative. No erection discomfort.

In preparing the patient for surgery, whether his injury is acute or subacute, any infection present must be controlled with adequate cleansing measures and antibiotics.[30] Tetanus antitoxin should be

Fig. 2a. J. T. 23 year old Negro farmer received burns of penis, legs and right arm when attempting to extinguish a gasoline fire on his tractor, resulting in loss of skin encircling penile shaft.

Fig. 2b. J. T. Six weeks following split-thickness skin graft. Patient states sensation has returned and he lives a satisfactory married life.

given routinely. If the accident presents itself to the surgeon early enough, there is a possibility that the wound may be cleansed, debrided, and resurfaced in one initial operation.[43] On the other hand, if the injury is chronic and grossly infected every precaution should be exercised in preparing the wound.[37]

Most cases involve only the integument of the penile shaft. The new skin covering should be characterized by close resemblance to the original covering in thickness, texture, and tactility. It should be relatively free of hair and exhibit a good cosmetic appearance. The donor site must be chosen with the above points in mind. The abdomen, lower chest, or inner surface of the thigh are suitable areas. If the surgeon decides to utilize a split-thickness skin graft, which has been the choice in the cases on our service, the skin is removed from a relatively hair-free area and superficially enough so that any hair follicles present may not be involved. Our technic is as follows:

The patient is placed upon the operating table in the dorsal recumbent position and under general anesthesia the donor and recipient sites are shaved, cleansed thoroughly and draped. A Foley retention catheter, utilized to empty the urinary bladder, is clamped and left in place. In our experience, it has been thought best to remove the granulation tissue present, exposing the superficial penile fascia but taking care to avoid penetration of this fascia. If the

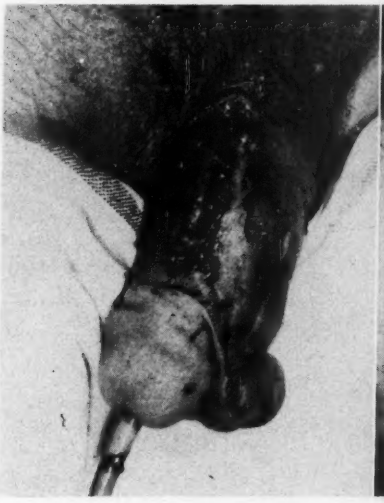


Fig. 3a. P. A. 55 year old tortilla-maker admitted to hospital with a urethral fistula and extravasation of urine into soft tissues of penis and scrotum, due to impacted calculi in penile urethra. Also presents severe chemical (drug) burn on skin of penis. Figure shows organ after one month of therapy.

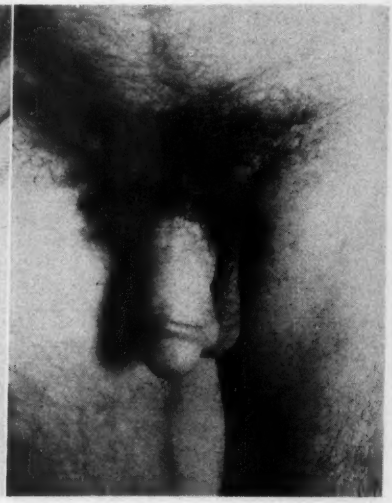


Fig. 3b. P. A. Six months after split-thickness skin graft was applied and urethral fistula closed.

correct plane has been exposed there is very little bleeding. The skin at the base of the penis is darted to prevent future constrictions. Hemostasis is easily accomplished with moist saline sponges and any sizable vessels are ligated with free hand ties of No. 40 stainless steel wire, which causes less tissue reaction and fewer complications in our hands.

From the donor area a one-piece split-thickness skin graft is removed and placed on a wet sponge with the raw surface exposed to simplify its application to the recipient site. The penile shaft is then straightened by tension on the indwelling catheter, the graft molded to its new host area and the moist sponge removed. The transplant is tailored to fit and then joined to the skin circling the penile base by interrupted sutures which are left long to act as stent ties. The opposite margin of the graft is similarly treated. The ends of the skin graft are approximated on the ventral aspect of the shaft, serrating or zig-zagging the line of closure to prevent any longitudinal constricting band which may restrict erection. The area beneath the skin graft is thoroughly irrigated with cool saline solution to rid the bed of any blood clots. It is not necessary to baste the graft along the shaft because after a dressing of fine mesh gauze and fluffs are placed over the graft the sutures that were left long are now tied over the dressing to produce a contour-fitting pressure bandage which obliterates any dead space. Several no. 36 stainless steel wire sutures are positioned in circular formation about 7.5 to 10 cm. from the penile base, and after addition of more cushion in the way of gauze fluffs, these sutures are tied over the entire bandage positioning the penile shaft perpendicular to its attach-

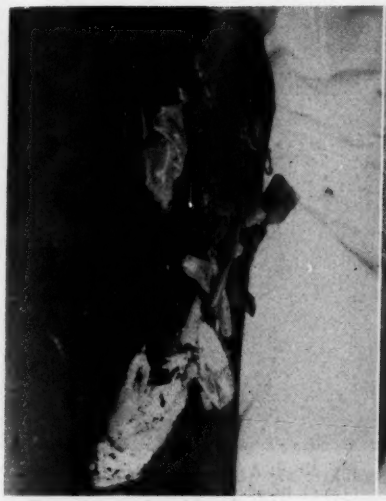


Fig. 4a. B. H. 66 year old Negro man was hospitalized because of a urinary extravasation and necrotizing infection due to a ruptured urethral stricture. The condition was cleaned up by incisions, drainage and antibiotics.

Fig. 4b. B. H. There remained an infected, denuded, edematous penis, which was treated by wet dressings and elevation for six weeks before it was ready for surgery.

Fig. 4c. B. H. Six days postoperative. A split-thickness skin graft was utilized.

ment on the abdominal wall. Elastoplast is utilized to protect the bandage and also to make a Bellevue bridge to support the scrotum. The catheter is opened and later connected to a bedside drainage bottle to prevent urinary contamina-

Fig. 5a. J. A., 40 year old Negro log cutter. Six months after gunshot avulsion of the penis, scrotum and testes.

Fig. 5b. J. A. Anterior view. Result of a tubed flap from abdomen.

Fig. 5c. J. A. Lateral view of the reconstructed phallus.

tion of dressings. The bandage is first changed on the fourth day and the catheter removed on the seventh day. Because of intermittent erections, there is a minimum of graft shrinkage. None of the patients have complained of tightness about the shaft and one patient has been aware of sensory restoration in six weeks (figs. 1-5).

There is a difference of opinion as to what should be done with remnants of skin remaining attached to the distal end of the penis at the corona.[1,5,8,9,10,20,32,39,43] If they are viable, these may be utilized; in our experience they fortunately have not developed edema. Bruner[9] reported a case in which he was able to approximate the rem-

nant preputial collar to the remaining pubic skin and scrotal tags and obtain an excellent result.

There are other methods of integumental restoration, such as a scrotal flap,[2,3,8,10,11,29,40,44,56,57] a tunnel skin graft or flap,[6,14,46,51] etc. Grafts are perhaps the most useful method.[4,13,18,20] Owens,[37] in an excellent and exhaustive review in 1942, presented advantages and disadvantages of both flaps and grafts, but he is satisfied that the latter are better for superficial losses of the penis. In a thorough study, May,[32] suggested that it is worthwhile to replace the skin of the penis if any is available because of its regenerative power; otherwise he feels that split skin grafts are also the method of choice.

When the entire penis is avulsed, a skin flap is usually the choice for reconstruction.[5,21,23,35] Figures 5a, 5b and 5c show an example in a Negro adult who lost his entire phallus and scrotal contents due to a shotgun injury. During the act of micturition, he found himself unable to guide the stream and was annoyed with urine running down his leg and on his clothes. To control this, he utilized a funnel, placing the wide end next to his perineum and the spout would focus the stream to its destination. We constructed a tubed flap which was advanced from the left abdomen to the pubic area. The next procedure will be to construct a urethra by means of the Dennis Browne technic and finally a resemblance of a filled scrotum.

The plan of approach for total or partial loss of the scrotal skin should be to form as soon as possible a protective covering for the testes in order to protect their function. Following subsidence of any existing infection, they then may be temporarily or permanently pocketed on their respective sides subcutaneously in the thigh,[6,41,47,54,56] the inguinal area, or the abdomen;[32] or covered by stretching the remaining remnant of scrotal tissue over the testes.[55] Skin flaps are usually constructed and utilized to form the sac for the testes. Scrotal tags or fragments remaining should always be saved because these can often be stretched enough to form an entirely satisfactory scrotum.[27,36,43,55] Split-thickness skin grafts are too constricting about the testes to be feasible.

Conclusion

In a review of the literature since 1855, approximately 84 cases of loss of the skin of the penis and scrotum have been reported. Though a sporadic problem for centuries, this type of injury is definitely on the increase because of the hazards of modern machinery and farm implements. Consideration of the psychological stress is of utmost importance. An understanding of the anatomy and physiology of the involved organs and adherence to basic surgical prin-

ciples will in most instances produce satisfactory functional and cosmetic results.

REFERENCES

1. Baxter, H.; Hoffman, M.; Smith, E., and Stern, K.: Complete avulsion of skin, penis, and scrotum; surgical, endocrinological and psychological treatment. Plast. & Reconstruct. Surg. *4:*508 (Nov.) 1949.
2. Bean, L. L.: Skin graft of penis, U. S. Nav. M. Bull. *47:*715 (July-Aug.) 1947.
3. Benedict, S. R.: Scrotal skin graft in severe injuries of penis, South. M. J. *11:*438 (June) 1918.
4. Bergman, R. T.; Howard, A. H., and Barnes, R. W.: Plastic reconstruction of penis, J. Urol. *59:*1174 (June) 1948.
5. Blum, V.: Case of plastic restoration of the penis, J. Mt. Sinai Hosp. *4:*506, 1937-38.
6. Bogoras, N. A.: Uber die volle plastiche Weiderherstellung eines sum Koitus fähigen Penis (Peniplastica totalis), Zentralbl. f. Chir. *63:*1271 (May 30) 1936.
7. Brown, J. B., and Muckenfuss, R. S.: Granuloma inguinale with report of case with oral involvement, Internat. J. Orthodontia *18:*988 (Sept.) 1932.
8. Brown, J. B.: Restoration of entire skin of penis, Surg., Gynec. & Obst. *65:*362 (Sept.) 1937.
9. Bruner, J. M.: Traumatic avulsion of male external genitalia, Plast. & Reconstruct. Surg. *6:*334 (Oct.) 1950.
10. Burford, C. E., quoted by Ehrich, W. S.[18]
11. Byrars, L. T.: Avulsion of scrotum and skin of penis; technic of delayed and immediate repair, Surg., Gynec. & Obst. *77:*326 (Sept.) 1943.
12. Carr, W. P.: Plastic operation for restoring scrotum; with report of case, Am. J. Dermat. & Genito-Urin. Dis. *15:*292, 1911.
13. Connell, W. K.: Operative treatment of elephantoid scrotum, Brit. J. Surg. *19:*651 (April) 1932.
14. Cottle, G. F.: Avulsion of scrotum, left testicle and sheath of penis, U. S. Nav. M. Bull. *20:*457 (April) 1924.
15. Counseller, V. S., and Palmer, B. M.: Avulsion of skin of penis and scrotum, S. Clin. North America *9:*993 (Aug.) 1929.
16. Cruickshank, M. M.: Denuded penis after operation for elephantoid scrotum, Indian M. Gaz. *72:*87 (Feb.) 1937.
17. Davis, A. D., and Brenner, R. E.: Primary repair of total avulsion of skin from penis and scrotum, Plast. & Reconstruct. Surg. *3:*417 (July) 1948.
18. Ehrich, W. S.: Two unusual penile injuries, J. Urol. *21:*239 (Feb.) 1929.
19. Esau, P.: Phlegmone und Grangrän an Penis und Scrotum, Arch. f. Klin. Chir. *122:*635, 1923.
20. Ferris, D. O.: Traumatic avulsion of skin of penis and scrotum, J. Urol. *62:*523 (Oct.) 1949.
21. Frumkin, A. P.: Reconstruction of male genitalia, Am. Rev. Soviet. Med. *2:*14 (Oct.) 1944.
22. Gibbs, R. W.: Case where entire scrotum and perineum together with one testicle and its cord attached and nearly all integument of penis were torn off; recovery with preservation of sexual powers, Charleston M. J. & Rev. *10:*145, 1855.
23. Gillies, H., and Harrison, R. J.: Congenital absence of penis, Brit. J. Plast. Surg. *1:*8 (April) 1948.
24. Greene, L. B.: A case of acute streptococcus penile gangrene, U. S. Nav. Bull. *18:*243 (Feb.) 1923.
25. Guitéras, R.: Plastic operation for new scrotum with report of case of gangrene and slough of integument over external genitals, J. Cutan. & Genito-Urin. Dis. *20:*211, 1902.
26. Hamm, W. G., and Kanthak, F. F.: Gangrene of penis following circumcision with high frequency current, South. M. J. *42:*657 (Aug.) 1949.
27. Jacobs, F. M., and Berry, J. L.: Reconstruction of scrotum; new use for prepuce, J. Urol. *61:*956 (May) 1949.

28. Judd, E. S., and Havens, F. Z.: Traumatic avulsion of skin of penis and scrotum, Am. J. Surg. *62:*246 (Nov.) 1943.

29. Koch, K.: Missglückter Versuch gestielter Plastik aus dem Skrotum, Zentralbl. f. Chir. *55:*587 (March 10) 1928.

30. Koch, S. L.: Injuries of parieties and extremities, Surg., Gynec. & Obst. *76:*1 (Jan.); 189 (Feb.) 1943.

31. König, Ernst: Plastischer Ersatz des Hodensaskes, Arch. f. Klin. Chir. *150:*244, 1928.

32. May, H.: Reconstruction of scrotum and skin of penis, Plast. & Reconstruct. Surg. *6:*134 (Aug.) 1950.

33. Meyer, H.: Phalloplastik nach Schindung, Arch. f. Orthop. *27:*437 (July) 1929.

34. MacDonald, J. A.: Plastic operation for complete loss of scrotum, Indiana M. J. *26:*227, 1907.

35. McIndoe, A.: Deformities of male urethra, Brit. J. Plast. Surg. *1:*29 (April) 1948.

36. Nielson, H. K.; Ferris, D. O., and Logan, G. B.: Injury of penis, scrotum and buttocks of newborn resulting in gangrene, Am. J. Dis. Child. *75:*85 (Jan.) 1948.

37. Owens, N.: Reconstruction for traumatic denudation of penis and scrotum, Surgery *12:*88 (July) 1942.

38. Pfeiffer, D. B., and Miller, D. B.: Traumatic avulsion of skin of penis and scrotum, Plast. & Reconstruct. Surg. *5:*520 (June) 1950.

39. Powers, C. A.: Complete denudation of penis, Ann. Surg. *49:*238, 1909.

40. Rihmer, B. von: Traumatische Entblössung des Penis; Phalloplastik, Ztschr. f. Urol. *26:*369, 1932.

41. Robertson, J. F.: Avulsion and reconstruction of scrotum, South. Med. & Surg. *93:*527, 1951.

42. Robinson, D. W.; Stephenson, K. L., and Padgett, E. C.: Loss of coverage of penis, scrotum and urethra, Plast. & Reconstruct. Surg. *1:*58 (July) 1946.

43. Roth, R. B., and Warren, K. W.: Traumatic avulsion of skin of penis and scrotum, J. Urol. *52:*162 (Aug.) 1944.

44. Sproat, S. M.: Unusually extensive injury, California & West. Med. *23:*1318 (Oct.) 1925.

45. Stirling, W. C.: Case of gangrene of scrotum and penis, J.A.M.A. *80:*622 (March 2) 1923.

46. Sutton, L. E.: Reconstruction following complete avulsion of skin of penis and scrotum, New York State J. Med. *43:*2279 (Dec. 1) 1943.

47. Thierry, W.: Ein Fall von Totalgangrän des Scrotums und der Penishant, München. Klin. Wchnschr. *66:*937, 1919.

48. Thorek, P., and Egel, P.: Reconstruction of penis with split-thickness skin graft, Plast. & Reconstruct. Surg. *4:*469 (Sept.) 1949.

49. Vermooten, V.: Use of estrogenic hormone in plastic operations on penis, J. Urol. *58:*391 (Nov.) 1947.

50. Vernon, H. R., and Kelly, R.: Unusual surgical emergency, Brit. M. J. *1:*925 (May 4) 1935.

51. Veseen, L. L., and O'Neil, C. P.: Plastic operation on penis, J. Urol. *30:*375 (Sept.) 1933.

52. Vodi, E.: Cirugía Plastica. Presentacion de un Casa, Bol. Asoc. Méd. de Puerto Rico *22:*25, 1929.

53. Welch, K.: Avulsion of skin of scrotum and penis, M. J. Australia *1:*527, 1918.

54. Westbrook, R. W.: Complete avulsion of integument of scrotum and penis, Long Island M. J. *5:*405, 1911.

55. Wetherell, F. S.: Loss of scrotal integument; simple method of repair, Surgery *18:*525 (Oct.) 1945.

56. Whelan, E. P.: Repair of avulsed scrotum, Surg., Gynec. & Obst. *78:*649 (June) 1944.

57. Whelan, E. P.: Reconstruction of penile urethra following trauma, Surg., Gynec. & Obst. *72:*81 (Jan.) 1941.

THE DEVELOPMENT OF SARCOMA IN BONE SUBJECTED TO IRRADIATION

A Case Report

RICHARD DE YOUNG, M.D.
Galveston

ATTENTION has recently been directed to the fact that sarcoma in bone occasionally occurs following roentgen or radium irradiation. Hatcher[2] reviewed the literature of the experimental production of sarcoma in bone by radium or roentgen radiation. He also reviewed the clinical cases of osteogenic sarcoma following irradiation, noting that there were reports in the literature of 24 patients who developed bone sarcoma following exposure to irradiation.

Hatcher added 3 cases of his own in which bone sarcoma developed in areas which were irradiated for other lesions.

Harrison S. Martland[3] reviewed the pathologic findings in the "Radium Dial Painters" and noted that there were four deaths from osteogenic sarcoma in seventeen deaths among the dial painters.

William G. Cahan and others,[1] in a comprehensive report reviewing the experimental and clinical literature, described 11 cases of sarcoma of bone occurring in areas 7 to 22 years after radiation. Four of their cases followed irradiation of microscopically proved benign lesions. Five followed irradiation of lesions diagnosed as benign by roentgenologic studies, and two occurred in bones which were radiologically normal at the time of irradiation. While the evidence that irradiation produced the sarcomatous lesions is not incontrovertible, the likelihood of its being responsible for the ensuing sarcomas is great.

Inasmuch as irradiation is known to produce leukemia in the myeloid tissue and is conducive to epithelioma in heavily irradiated skin, it would appear to be reasonable that the sarcomas which develop in bone following heavy irradiation are a comparable reaction.

CASE REPORT

A. D. P., a 12 year old white boy, was first seen in the Orthopedic Clinic of John Sealy Hospital in January, 1946, complaining of shortening and knock knee of the left leg. The history revealed that at the age of 3 years he had suffered from a mild attack of anterior poliomyelitis which left some residual

From the Department of Surgery, Orthopedic Division, University of Texas School of Medicine, Galveston.

weakness of the left lower extremity. At 9 years of age, the patient had a release of the iliotibial band at the left knee joint for a developing genu valgus. At the time he was seen in the clinic, in January, 1946, there was shortening of three-fourths of an inch in the left lower extremity and genu valgus of this extremity.

Fig. 1. Roentgenogram taken June 25, 1948, shows osteolytic process in metaphyseal region. At time of roentgenogram this was not considered significant.

Fig. 2. Roentgenogram taken August 15, 1950, shows the sarcoma in same area as previously noted osteolysis.

It was decided to give the patient irradiation of the right distal femoral epiphyseal plate to retard the growth of the right lower extremity. This was carried out in July of 1945, the patient receiving 3,200 r. units in this region. Roentgenograms taken of these extremities showed no abnormality of the bones of the lower extremity except a genu valgus of the left leg.

The patient returned in June, 1947, at which time measurements revealed the same leg-length discrepancy. Again, the right distal femoral epiphyseal plate was irradiated, this time receiving 600 r. per day for six days for a total of 3,600 r.

The patient was seen in the clinic several times during 1948 and 1949, at which times roentgenograms of the lower extremities showed no abnormalities except the genu valgus and continued shortness of the left lower extremity (fig. 1).

In August, 1950, the patient was seen in the Orthopedic Clinic complaining of a painful tumor on the medial aspect of the right femoral distal metaphysis which had been present for two weeks. Roentgenograms taken at this time showed a destructive proliferative bone lesion suggestive of osteogenic sarcoma. He was admitted to the hospital for a more extensive work-up and for biopsy section. Roentgenologic bone survey taken at this time was negative. Examination revealed a healthy 16 year old white boy with a palpable tumor 4 by 6

cm., not adherent to the skin, on the medial aspect of the right distal thigh just above the knee. There was an 8 cm. scar along the anterolateral aspect of the left distal thigh just proximal to the joint. The remainder of the examination was negative (fig. 2).

The urinalysis showed a specific gravity of 1.028; reaction, acid; albumin, none; sugar, none; red cells, none; white cells, few; and no casts.

Results of the Kolmer and Kahn tests were negative.

The hemoglobin was 13.6 Gm. per 100 cc.; erythrocytes, 3,900,000 per cu. mm.; the leukocytes 7,850 per cu. mm.; with the differential as follows: neutrophiles, 58; lymphocytes, 41; basophile, 1; slight microcytosis.

On August 16, 1950, a section of the lesion of the right distal femoral metaphysis was obtained which on frozen section showed evidence of malignancy. While the tourniquet remained on the thigh, the patient was taken to the Radiology Department where he received 2,480 r. before the tourniquet was released. He subsequently received during the following seven days a total of 12,400 r. to the region of the tumor.

On August 28, a disarticulation of the hip was done.

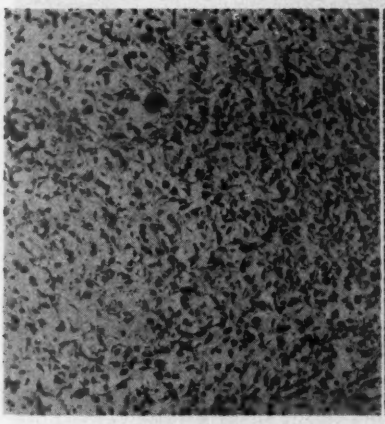


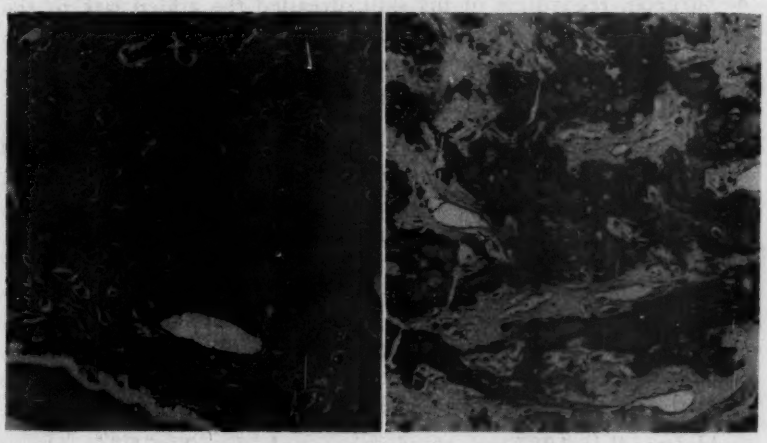
Fig. 3. Mitotic figures, hyperchromatic nuclei, and general pleomorphism characterize this section.

Fig. 4. The darkly stained areas are osteoid tissue.

Examination of the fixed section revealed marked pleomorphism in the cellular areas (fig. 3). There were bizarre shaped cells dispersed throughout a faintly acidophilic staining matrix. There were four to five mitotic figures per high power field. The cells had a narrow rim of cytoplasm surrounding the hyperchromatic nuclei. The vessels were rather prominent throughout the sections. At some points, the cells aligned themselves along the acidophilic staining matrix or osteoid tissue (fig. 4). At other points, the cells assumed a spindle shape and were arranged very compactly in intertwining bundles. Mitotic figures were equally prominent in these areas. Still other sections of the tumor showed a predominance of cartilage. The cartilage cells were distributed irregularly throughout this matrix and showed a marked variation in size and shape. The cartilage and osteoid elements were arranged in haphazard manner without any suggestions of uniformity.

A diagnosis of osteosarcoma was made.

The prepared slides from the specimen obtained at amputation showed osteosarcoma with marked radiation reaction. In contrast to the original biopsy section, there were practically no mitoses observed in these sections; however, viable tumor cells were remaining among the necrotic tumor and bone (figs. 5 and 6).



Figs. 5 and 6. These sections show the reaction of the tumor to preoperative irradiation. Viable tumor cells are still present.

At the time he was last seen in the Orthopedic Clinic on August 7, 1951, there was roentgenologic evidence of pulmonary metastasis. The patient died on January 4, 1952.

Comment. This case is particularly interesting inasmuch as an osteogenic sarcoma developed in the femoral metaphysis of a white boy while he was under observation. Roentgenograms of the region in which the sarcoma arose were obtained at yearly intervals for four years prior to the development of the sarcoma. In none of these roentgenograms was there detected any evidence of abnormality of the metaphysis, the region in which the sarcoma developed, until the patient presented himself with a tumor in this region.

While the evidence that the irradiation was responsible for the development of sarcoma in this case is not incontrovertible, it is most suggestive.

REFERENCES

1. Cahan, W. G., and others: Sarcoma arising in irradiated bone, Cancer *1:*3 (May) 1948.
2. Hatcher, C. H.: Development of sarcoma in bone subjected to roentgen or radium irradiation, J. Bone & Joint Surg. *27:*179 (April) 1945.
3. Martland, H. S.: Occurrence of malignancy in radioactive persons, Am. J. Cancer *15:*2435 (Oct.) 1931.

INTESTINAL ANTISEPSIS

FRED J. WOLMA, JR., M.D.
Galveston

PRIOR to the advent of modern-day intestinal antiseptics, every surgeon, regardless of his skill, dreaded the added risk to the patient of opening the bowel and exposing the peritoneal cavity to its contents. When circumstances necessitated opening the bowel, great care was taken to prevent gross contamination. Bowel resection and suture by the "closed" or aseptic technic was mandatory to reduce contamination to a minimum. The "open" technic of resection and suture, even though sutures could be placed more accurately, was usually prohibited because of the fear of contamination which often resulted in sepsis and peritonitis followed by death. With modern-day antibiotics and intestinal antiseptics the "open" technic employing exacting and accurate suture is used by many surgeons in many clinics throughout the country. It should be emphasized, however, that even though intestinal antiseptics are used, sound surgical judgment and careful surgical technic should not be altered or sacrificed to any degree.

Halstead[1] in 1887 was the first to emphasize the importance of the submucosal suture without penetrating into the lumen of the bowel. This surgical technic fostered an enduring tight closure with security against infection and abscess formation and possible peritonitis.

The problem of intestinal antisepsis has been of interest to physicians and surgeons for at least 75 years. Furthermore, it has also been demonstrated in man that the diet of the adult has little effect on the qualitative bacterial population. Marshall[2] and his co-workers showed that coliform bacteria in the feces of mice could be reduced significantly by the administration of sulfanilylguanidine; however, the effect could not be produced in dogs even when the drug was administered in toxic doses over a prolonged period of time.

In 1942 Poth et al.[3] introduced succinylsulfathiazole (sulfasuxidine) as an intestinal antiseptic. In 1943 Poth and Ross[4] described several other sulfonamides, including phthalylsulfathiazole (sulfathaladine), which possessed local antibacterial activity.

Streptomycin has been used as an intestinal antiseptic. It is very effective against *alpha streptococcus fecalis;* however, many bacteria rapidly establish a flora resistant to this antibiotic.

From the Department of Surgery, University of Texas Medical Branch, Galveston.

Other agents used as intestinal antiseptics are aureomycin, bacitracin, chloromycetin, and neomycin. These agents will be discussed in more detail subsequently.

There are certain properties an ideal practical intestinal antiseptic must possess. Poth[7] has listed these properties as follows:

1. A low toxicity for the patient.
2. A broad bacterial spectrum.
3. Prevents development and overgrowth of resistant bacterial variants.
4. Rapid activity.
5. Limited absorption from the gastrointestinal tract.
6. Permits adequate food and fluid intake.
7. Assists in mechanical cleansing of the bowel without causing dehydration.
8. Will not cause gastroenteritis.
9. Promotes tissue healing.
10. Low dosage.
11. Soluble in water.
12. Palatable.
13. Does not interfere with tissue growth and repair.
14. Will inhibit the excessive growth of fungi.

Agents Used as Intestinal Antiseptics

The effective antibacterial agents used in the gastrointestinal tract may be divided into two main groups, namely: A., Bacteriostatics and B., Bactericidals.

A. *Bacteriostatic Agents.* The bacteriostatic group is composed of the sulfa drugs which include sulfasuxidine, sulfathaladine, and phthalysulfacetamide. There are certain organisms in the gastrointestinal tract which none of the sulfa drugs will inhibit: *i.e.*, the *alpha streptococcus fecalis,* which is always present in large numbers; however, this organism is ordinarily nonpathogenic. The retention of certain nonpathogenic bacteria in the gastrointestinal tract may be advantageous towards preventing the outgrowth of undesirable higher plant forms, *i.e.*, fungi and yeasts.

Sulfasuxidine and sulfathaladine are agents widely used for preparing the gastrointestinal tract for surgery. If used properly, these agents meet well the requirements of a practical intestinal antiseptic. The agents are inexpensive, nontoxic, and patients may be prepared either on an in-patient basis or an out-patient basis. Since it is the

policy of the University of Texas to work up all elective surgical cases as out-patients, these patients which are candidates for bowel surgery are placed on the drug while still out-patients awaiting completion of their work-up and admission to the hospital. After admission to the hospital, no time is lost preparing the bowel.

When employing sulfasuxidine and sulfathaladine as intestinal antiseptics, it is recommended that the following regime be followed: Sulfasuxidine is given in doses of 3 Gm. every four hours around the clock. The question arises in the minds of many: "Is it necessary to take the 12 o'clock midnight and the 4 o'clock a.m. doses in view of the inconvenience caused to the patient?" The answer to this question is definitely, "yes." If one remembers the action of these agents, *i.e.,* they are bacteriostatic in their action and depend on constant contact with the organisms of the gastrointestinal tract for their action, the reason becomes obvious. If the drug is not given continuously, regrowth of the organisms occurs during the eight hours no drug is given if the 12 o'clock midnight and the 4 o'clock a.m. doses are omitted, with the result that the total bacterial count of the stool is not considerably reduced. The patient is also placed on a low residue diet. A low residue diet is necessary because it not only aids in mechanical cleansing of the bowel, but because of the type of residue formed in the colon, more drug is allowed to come into direct contact with a larger surface of residue, thus resulting in a lower total bacterial count in the stools. Seven days are required to eliminate the pathogens from the intestinal flora. It is a common policy for individuals to attempt to prepare the bowel in three to five days. Repeated stool examinations have proved the ineffectiveness of this method. Sulfathaladine is used in doses of 1.5 Gm. every four hours only if the patient develops diarrhea while on sulfasuxidine. Sulfathaladine produces a ropy, soft, sticky type of stool, therefore it will prevent the mechanical cleansing of the bowel and should not be used routinely unless diarrhea is present. It is important to bear in mind that cleansing enemas and harsh purgatives are not required to cleanse the bowel preoperatively. These only lead to undesirable dehydration of the patient. The antibacterial agent should mechanically cleanse the bowel without producing dehydration. If the patient has an obstructing lesion of the colon and a first stage decompressing colostomy has been resorted to as an emergency procedure, the patient may be prepared for definitive surgery by using the agent orally and by introducing an equal quantity of the sulfa drug into the distal loop of the colostomy. Occasionally the distal colon may require irrigation with a mixture of sulfasuxidine and soda bicarbonate.

Postoperatively as soon as the patient is placed on per oral feed-

ings sulfathaladine is administered 1.5 Gm. every four hours. The patient is kept on a low residue diet. The agent is continued for 12 days postoperatively if there is any danger of disruption of the bowel sutures. Poth[6] in 1948 demonstrated clearly the favorable influence of intestinal antiseptics on bowel healing. The use of sulfasuxidine and sulfathaladine employed in the above manner, and in conjunction with good surgical technic and sound surgical judgment will yield excellent results in gastrointestinal surgery.

B. *Bactericidal Agents.* The bactericidal group includes several antibiotics. The bactericidal agents are of greater potential value than the bacteriostatic agents because by the administration of the former group, the organisms can be eliminated from the gastrointestinal tract much more rapidly. In addition, the bactericidal agents destroy the viable organisms, whereas the bacteriostatic agents inhibit the growth and depend upon mechanical removal or natural death of the viable organisms residing in the bowel when treatment is begun.

1. *Penicillin* has no antibacterial activity as far as the gastrointestinal flora is concerned; however, it has been found that penicillin antagonized the action of sulfathaladine although it did not antagonize the antibacterial action of sulfasuxidine.[5]

2. *Streptomycin* was one of the first antibiotics studied. It was found to be rapid in its action to eliminate *alpha str. fecalis* as well as other bacteria in the gastrointestinal tract, but unfortunately resistant forms grew out very rapidly.

3. *Bacitracin* is of little value.

4. *Aureomycin* was found to be very effective in eliminating most bacteria from the gastrointestinal tract; however, the side reactions proved highly undesirable. It caused nausea and vomiting too frequently in patients being prepared for gastrointestinal surgery. This side reaction predisposes to unnecessary dehydration of the patient. Monilia infections about the anus and mouth, and in other portions of the intestine have been known to follow the administration of aureomycin.

5. *Terramycin* is unsatisfactory because it causes a bloody diarrhea and the development of ulcers in the gastrointestinal tract.

6. *Neomycin* as used on the surgical service of the University of Texas has been found to be the most effective antibiotic in altering the bacterial flora of the bowel. Neomycin, when administered orally, is only poorly absorbed from the gastrointestinal tract. However, when given parenterally it is sufficiently toxic to preclude

its use in this manner. To date over 350 patients in the University of Texas Hospitals have been administered this antibiotic by mouth and not one single toxic manifestation has been encountered. Neomycin is extremely rapid in its action; in most cases the bacteria were completely eliminated from the gastrointestinal tract within 24 hours. Following the elimination of bacteria, yeast grew out in large numbers; however, not one instance of yeast infection was noted. Neomycin does fail in that it does not always inhibit the growth of *Aerobacter aerogenes*. This observation led to the combined use of neomycin with sulfathaladine because sulfathaladine is consistently effective against this particular organism. The therapeutic schedule of the neomycin-sulfathaladine combination has proved successful on 154 patients.

Neomycin may be used alone to sterilize the bowel rapidly in emergency gastrointestinal surgery, or in nonobstructed cases. The length of time required to prepare the nonobstructed bowel depends on how rapidly the bowel may be mechanically cleansed, usually 24 hours.

For elective gastrointestinal surgery in the absence of obstruction and other contraindications, the following schedule is recommended:

(a) Low residue diet.

(b) A single dose of 30 to 60 cc. of castor oil is given; 1.0 Gm. of neomycin sulphate together with 1.5 Gm. of sulfathaladine is given orally at the same time the castor oil is given. Neomycin 1.0 Gm. and 1.5 Gm. of sulfathaladine are given every hour for 3 doses. Then the 1.0 Gm. of neomycin and 1.5 Gm. of sulfathaladine are given at four hour intervals until time of surgery. By the end of 24 hours the gastrointestinal tract is prepared if no obstruction is present. With the neomycin and sulfathaladine combination used in the above manner sterilization of the gastrointestinal tract is no longer a problem. The mechanical cleansing of the bowel is the only problem. The length of time required to prepare the bowel depends on the time required to mechanically cleanse the bowel.

(c) Usually within 48 hours postoperatively 1.5 Gm. of sulfathaladine is given on a four hour schedule if there is reason for postoperative medication.

Because of the rapid bactericidal action of neomycin to sterilize feces, the agent was used in acute gastrointestinal emergencies. The use of neomycin in emergency abdominal surgery was reported by Poth[8] *et al.* in 1951.

Neomycin is not toxic when administered by mouth in the prescribed dose. No tissue damage in the gastrointestinal tract has been observed. Nausea and vomiting does not accompany the ad-

ministration of neomycin. Neomycin also promotes primary healing of the suture line in intestinal anastomosis. Careful studies on patients receiving both neomycin alone and in combination with sulfathaladine, even for periods as long as six weeks, have failed to show evidence that bleeding, clotting, and prothrombin times have been prolonged in the slightest.

Discussion

The present-day methods of intestinal antisepsis have been presented. It is realized, however, that the material represents a review of the subject and not new or original work.

The exact evaluation of intestinal antiseptics in regard to their ability to reduce mortality and morbidity rates in gastrointestinal surgery is impossible. The complications of breakdown of the suture line resulting in fistula, abscess formation, peritonitis, sepsis and death have been reduced markedly since 1941 when the first effective intestinal antiseptics were introduced.

The sulfonamide intestinal antiseptics, which are bacteriostatic in their action, are practical agents for reducing the gastrointestinal flora when administered in the prescribed manner. Among the antibiotics used as intestinal antiseptics, neomycin seems to be the most effective, especially when combined with sulfathaladine. Because neomycin is not available for general use as yet, the sulfonamides serve well and are the agents of choice at the present time.

Above all, it must be borne in mind that no agent is a substitute for sound surgical judgment and careful surgical technic. When intestinal antiseptics are combined with good judgment and careful technic, results will be uniformly good.

References

1. Halstead, W. S.: Circular suture of intestine—experimental study, Am. J. M. Sci. *94:*436, 1887.
2. Marshall, E. K., Jr.; Bratton, A. C.; White, H. J., and Litchfield, J. T., Jr.: Sulfanilylguanidine; chemotherapeutic agent for intestinal infections, Bull. Johns Hosp. *67:*163 (Sept.) 1940.
3. Poth, E. J.; Knotts, F. J.; Lee, J. T., and Unui, F.: Bacteriostatic properties of sulfanilamide and some of its derivatives: 1. Succinylsulfathiazole, new chemotherapeutic agent locally active in gastrointestinal tract, Arch. Surg. *44:*187 (Feb.) 1942.
4. Poth, E. J., and Ross, C. A.: Bacteriostatic properties of sulfanilamide and some of its derivatives: III. Phthalylsulfathiazole, new chemotherapeutic agent locally active in gastroenteric tract, Texas Rep. Biol. & Med. *1:*345, 1943.
5. Poth, E. J.; Wise, R. I., and Slattery, M. P.: Penicillin-phthalylsulfathiazole antagonism, Surgery *20:*147 (July) 1946.
6. Poth, E. J.: Bowel healing as influenced by intestinal antiseptics, South. M. J. *41:*672 (Aug.) 1948.
7. Poth, E. J.; Fromm, S. M.; Wise, R. I., and Hsiang, C. M.: Neomycin, new intestinal antiseptic, Texas Rep. Biol. & Med. *8:*353, 1950.
8. Poth, E. J., and others: Critical analysis of neomycin as intestinal antiseptic, Texas Rep. Biol. & Med. *9:*631, 1951.

MECKEL'S DIVERTICULUM

Case Report of Patent Omphalomesenteric Duct

JOHN M. THIEL, M.D.
Galveston

PATENT vitelline or omphalomesenteric duct, representing a type of Meckel's diverticulum, is a very uncommon finding and relatively few have been reported in the surgical literature. The usual type of Meckel's diverticulum, on the contrary, has been reported on numerous occasions and was described by various observers long before the advent of modern surgery.

Probably the first description of a diverticulum of the small intestine was given by Fabricius Hildanus[7] in 1598 long before Meckel in 1808 described the diverticulum known by his name. Following Hildanus' description of an intestinal diverticulum, Lavater[12] in 1672 reported a similar one. In 1700, Littre[13] described a diverticulum of the ileum contained within a hernial sac, known since then as Littre's hernia, and in 1701 similar hernias were reported by Mery[16] and by von Taignon.[23] In 1707 Fredericus Ruysch[19] also described a diverticulum of the ileum and later, in 1769, Morgagni[17] described this diverticulum and suggested a congenital origin of diverticulum of the ileum, but it was not until 1808 that Meckel,[14] in the first of a series of three articles and subsequently in two other articles written between 1808 and 1815, described and discussed the diverticulum of the ileum now known by his name, as regards its embryology and clinical significance.

Most authors give the incidence of Meckel's diverticulum as between 1 and 3 per cent. According to Ladd and Gross,[11] Meckel's diverticulum occurs in 2 to 3 per cent of individuals coming to autopsy examination. Cole and Elman[3] give the incidence as 2 per cent of individuals. Harbin[6] in a series of 507 consecutive laparotomies in which Meckel's diverticulum was looked for found an incidence of 1.3 per cent. Christi[1] gives an incidence of 1.1 per cent in a series of 5,768 unselected autopsies. This percentage is less in other reports. Noel[18] found a 0.21 per cent incidence in about 12,000 intra-abdominal operations, a total of 25 cases being found. In 5,000 postmortem examinations done on fetuses and infants at the Chicago Lying-In Hospital of the University of Chicago, in a five year period only 10 cases of Meckel's diverticulum were found, an incidence of 0.5 per cent. Howell[8] in a 10 year period from 1934 to 1944 at Duke University gives an in-

From the Department of Surgery, University of Texas Medical Branch, Galveston, Texas. Read before the Texas Surgical Society, April 2-3, 1951, Galveston, Texas.

cidence of .05 per cent in 122,490 admissions, there being 61 cases of Meckel's diverticula. In 3,522 autopsies, Meckel's diverticulum was found three times giving an incidence of .08 per cent. In the John Sealy Hospital there are only 15 cases on record during the 10 year period from May 1941 to May 1951.

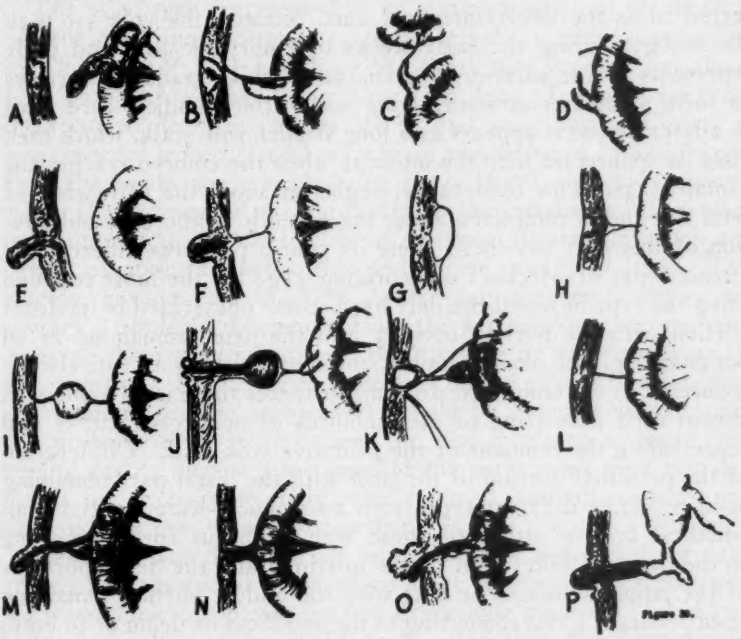


Fig. 1. Different variations of the normal development of the omphalomesenteric duct. *A, B, C* and *D* are types in which the proximal part has failed to undergo obliteration. Distal remains of the omphalomeseneric duct are shown in *E, F, G* and *H. I, J* and *K* are forms in which varying degrees of obliteration have occurred. The remaining drawings are variations in which the omphalomesenteric duct has persisted throughout its entire course.

(Reprinted from Kittle, Jenkins and Dragstedt[10] with permission of the authors and publishers.)

Meckel's diverticulum may be found at any age, but by far the majority of those causing symptoms are discovered in childhood. Of the 15 cases studied here 10 were in adults and only 5 in infants and children, but of these, only 2 of the 10 in the adults caused symptoms whereas 3 of the 5 in infants and children produced symptoms. They are found according to Howell[8] three times more frequently in males than in females. Curd[5] in a series of 670 cases found the occurrence 3.1 times greater in males than in females. Other authors agree on the incidence of the disease being more common in males, but the exact reason for the predominance in males has not been explained. In our series the ratio is

about equal, 8 males to 7 females, the incidence in the adults being 5 males and 5 females, and in the infants and children the ratio being 3 males to 2 females.

Meckel's diverticulum is the result of an incomplete obliteration of the vitelline or omphalomesenteric duct. This duct, also referred to as the vitello-intestinal duct, connects the yolk sac with the mid-gut during the early weeks of embryonic life, and early represents a wide anterior communication which gradually narrows to form a tubelike structure lying within the umbilical cord until at a later period it appears as a long slender yolk stalk, which then loses its connection with the intestine when the embryo reaches the 7 mm. stage. This obliteration begins in about the fifth week of fetal life and is completed about the seventh. Failure of obliteration of this duct anywhere along its course produces different anatomic types of Meckel's diverticulum (fig. 1), the most common being the type in which the duct has become obliterated in its distal portion with the portion opening into the ileum remaining as an out-pouching. In most instances this out-pouching is entirely unconnected to the umbilicus. In some instances there is an associated fibrous cord connecting to the umbilicus or nearby structures and representing the remnant of the primitive yolk stalk. Obliteration of the proximal portion of the duct with the distal part remaining open produces different types such as a mucus-secreting tract, an umbilical cyst or either of these with a fibrous cord connecting to the ileum. Obliteration of the proximal and the distal portions of the omphalomesenteric duct with the middle portion remaining open produces a cyst connecting to the umbilicus or ileum or to both. When the middle portion closes and the proximal and distal parts remain unobliterated a diverticulum from the ileum and a fistulous opening to the umbilicus or an umbilical cyst may result. A very uncommon type is that in which the entire portion of the vitelline duct remains unobliterated forming a fistulous connection between the ileum and the umbilicus and is the type reported in this paper. This is a very unusual type and in Ladd and Gross's[11] series of 73 patients only 1 case was of this type. Kittle, Jenkins and Dragstedt[10] found only 2 such cases in over 30,000 births at the Chicago Lying-In Hospital of the University of Chicago in a 10 year period preceding 1947 and in a survey of the literature they found only 128 reported cases. One other case was collected elsewhere making together with their 2, a total of 131 known cases of patent omphalomesenteric duct. Of the cases of Meckel's diverticulum recorded at the John Sealy Hospital, the case reported here is the only one of this type. However, 1 case not included in this series of Meckel's diverticula was reported from John Sealy Hospital by Singleton

and King[21] in February 1951. This was a 5 year old Negro boy in whom the end of appendix formed a fistulous opening at the umbilicus. Whether this represented a true patent omphalomesenteric duct is debatable, and the authors were unable to determine this with certainty.

The usual type represented by an outpouching from the ileum is present in 82.5 per cent of all cases of Meckel's diverticula according to Thompson.[22] The length of the usual type varies from about 1 to 7 cm, the average being 3 to 5 cm. long. It may arise from the terminal ileum in various places, and according to Christi[1] it was observed to occur in 63 cases in an area between 15 and 90 cm. from the cecum, the average being about 50 cm. Meckel's diverticulum is composed of all the layers of the intestinal wall in contradistinction to all other types of intestinal diverticula which are devoid of muscular layers, thin walled and usually multiple. The mucosa, however, of Meckel's diverticulum frequently shows important pathologic differences from the surrounding ileal mucosa, many of them seeming to have the presence of gastric mucosa in some portion. In our series, however, only 1 case was mentioned as containing gastric mucosa. It should be noted, however, that this probably does not represent the true incidence of those containing gastric mucosa since some of the early cases were not sectioned for microscopic study after removal, and others were not removed for one reason or another. In one instance a Meckel's diverticulum was the cause of intussusception with resulting gangrene of a segment of ileum which after resection apparently was not studied microscopically because of its poor condition. Ladd and Gross [11] in their series of 73 cases noted that those containing gastric mucosa represented the largest percentage, this type being present in 40 cases as compared with 24 cases containing ileal mucosa only. In their series there were 4 cases of duodenal and ileal mucosa, 4 cases of colonic and ileal mucosa and 1 case containing pancreatic tissue and ileal mucosa. The predominance of gastric and ileal mucosa in these diverticula is borne out by other authors. Schaetz[20] found it in 16.6 per cent of 37 specimens, and Hudson and Koplik[9] found gastric mucosa present in 52 per cent of 23 specimens.

It is not the intent of this paper to give in detail the symptoms and findings of Meckel's diverticulum since these are adequately presented in many articles on the subject. Suffice to say that for the most part they depend upon the various complications which may develop. In many instances, patients having a Meckel's diverticulum are completely asymptomatic while others have various symptoms and findings, the most common being due to hemorrhage from

some point within the diverticulum. Various authors confirm the high incidence of bleeding whereas in our series only 2 patients are reported as having evidence of bleeding. The bleeding usually is caused by ulceration which when present occurs mostly at the juncture of gastric and ileal mucosa at the base of the diverticulum. Perforation is also a common complication of ulceration in this area and produces the usual symptoms associated with a leakage from a hollow viscus. Cobb[2] reported 110 cases of ulceration in Meckel's diverticula associated with the presence of gastric mucosa and gave an incidence of 72 per cent with hemorrhage and 55 per cent with perforation. Perforation as well as bleeding is apparently most commonly noticed in children and is relatively uncommon in the older age group. Ladd and Gross[11] in their series of cases list hemorrhage as having occurred in 26, intussception in 17, abdominal pain in 12, inflammation with or without perforation in 10, obstruction from a band to the umbilicus in 6, umbilical fistula in 1 case and volvulus and infarction of the diverticulum in 1 other case. Other authors report additional complications such as herniation, malignancy arising from within Meckel's diverticulum, calculi contained within the diverticulum, and others. Of the 15 cases in the past decade at the John Sealy Hospital 7 were incidentally found and resected at operations for other conditions and apparently were asymptomatic; 2 were incidentally found at operations for other conditions, had wide bases, were thought to be uninvolved and asymptomatic and were not removed; 1 was found at autopsy and apparently had been asymptomatic. It is interesting to note that of the 10 apparently symptomless diverticula, all occurred in adults except 2, whereas of the 5 cases having symptoms and findings definitely attributable to Meckel's diverticula, 3 were in infants and young children and 2 were in young adults. One such case was diagnosed by barium roentgenologic studies in a 7 year old girl in whom occult blood was found in the stool and who had a history of passage of "dark blood" following an enema on one occasion, and of having episodes of lower abdominal pain associated with fever and vomiting. This patient, however, was removed from the hospital without operation. One other patient, a 22 year old woman had vague abdominal pain in the right lower quadrant of the abdomen of two months' duration. At operation the appendix was grossly normal; a Meckel's diverticulum was found and was resected. Still another patient, a 36 year old woman was found to have an acute Meckel's diverticulitis with acute peritonitis complicating an eight months' pregnancy. This patient was in labor, had a rigid abdomen and was thought to have a ruptured uterus since she has been delivered of her last baby by Caesarean section. One Meckel's diverticulum in a 2

year old boy was the leading point of an intussusception with resulting gangrene of the terminal ileum, necessitating resection of the involved portion. One case in a newborn male infant, of complete communication between the ileum and the umbilicus with a large external fistulous opening at the umbilicus is reported in this paper.

Case Report

This patient, a well developed, well nourished full term male infant was born by normal delivery on Dec. 28, 1950 in the John Sealy Hospital. Apparently nothing abnormal was noticed and the patient was discharged on

Fig. 2. Lateral view showing protrusion of omphalomesenteric duct at umbilicus.

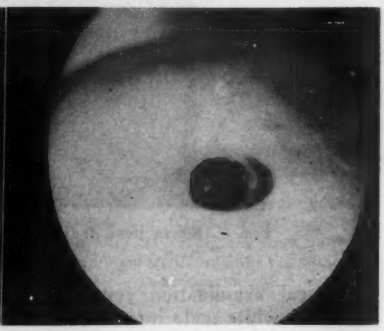


Fig. 3. View looking down upon external opening of patent omphalomesenteric duct.

Fig. 4. Roentgenogram, lateral view, after injection of Lipiodol through catheter into external opening of omphalomesenteric duct at umbilicus, showing the Lipiodol in the small intestine.

Fig. 5. Oblique roentgenogram view showing Lipiodol in small intestine.

December 31, three days after birth. He was readmitted to the hospital on Jan. 9, 1951 and the history obtained from his mother at that time was that she had noticed that the stump of the cord had seemed quite large and that on Jan. 5, 1951, eight days after birth, the umbilical cord dropped off leaving a reddish, moist stump at the umbilicus. Following this the mother noticed

the escape of gas bubbles from the umbilical stump and three days after the cord had dropped off there was a red, cylindrical protrusion from the center of the stump which persisted and at the time of admission was about twice as long as when first noticed. No fecal drainage from the umbilical stump was noticed until the day of admission when the passage of a small amount of yellow feces occurred in the Emergency Room. The stools apparently had been normal since birth and had been four to five in number per day. The patient vomited twice the day before admission.

Fig. 6. Subsequent drainage film showing Lipiodol in colon.

Physical examination revealed an apparently well developed and well nourished white male infant 12 days of age. The abdomen was slightly distended and there was a light red, moist protrusion about the size and shape of a strawberry extending through the umbilicus and apparently covered by intestinal mucosa (figs. 2 and 3). The skin of the abdomen was irritated and there were numerous papulo-pustular lesions over the diaper area. Roentgenologic studies showed some dilated large and small bowel with no definite air fluid levels. There was nothing diagnostic about these films, but after injection of Lipiodol through the external opening in the umbilicus, it was found to enter the small bowel (figs. 4 and 5). Subsequent drainage films showed all the Lipiodol to be in the colon (fig. 6). The diagnosis of patent omphalomesenteric duct was thus substantiated and the patient was prepared for operation.

This was done on Jan. 15, 1951, the eighteenth day after birth (fig. 7). An incision was made to encircle the protruding bowel wall and was extended transversely through the skin on the right for a distance of about 1 inch and on the left for about ½ inch. The umbilical portion of the diverticulum was dissected free from the abdominal wall and the entire diverticulum delivered (fig. 8). It was found to be about 5 cm. in length and was connected to the terminal ileum 13 inches from the ileocecal valve. A fibrous cord representing the obliterated vitello-intestinal artery was present along the superior surface of the duct. Oblique resection of the diverticulum was done and the defect in the ileum was sutured obliquely in two layers, No. 0000 plain catgut continuous suture being used for the inner row and No. 0000 interrupted braided black silk for the outer row (fig. 9). The appendix measuring 5 cm. in length was also removed. The abdominal wall at the site of the fistulous opening was sutured and the umbilicus was reconstructed. Examination of the gross specimen postoperatively showed it to include the

umbilical portion measuring 2½ by 1½ cm. attached to a tubelike section 1½ cm. in length (fig. 10). Microscopic section of the specimen showed it to be lined with squamous epithelium on one side and intestinal epithelium on the other. Further study of the intestinal mucosa revealed branching deep gland formation showing a number of goblet cells and mild chronic inflammatory reaction in the stroma (figs. 11 and 12). The appendix showed no abnormality. The postoperative course was uneventful and the patient was discharged from the hospital on Jan. 27, 1951 (fig. 13).

Fig. 7. Appearance at time of operation, four days after pictures seen in figures 2 and 3 were taken. Note increased protrusion and eversion of mucosa covered wall of omphalomesenteric duct.

Fig. 8. Appearance at operation after dissection and freeing from umbilical region and delivering the duct and associated loop of ileum into the abdominal incision.

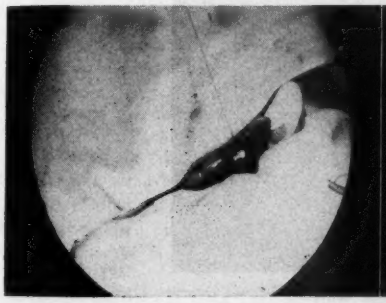


Fig. 9. Ileum at site of resection of the patent omphalomesenteric duct, showing appearance at completion of two layer repair.

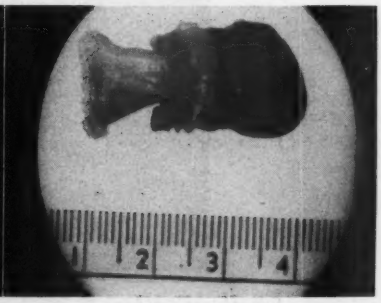


Fig. 10. Postoperative gross specimen showing line of resection from ileum, attachment at umbilicus, and eversion of duct through external opening.

Comments. This case is a rare type of Meckel's diverticulum in which the entire length of the omphalomesenteric duct was patent and had not become obliterated, and it presents the typical history, and the physical and roentgenologic findings of the uncomplicated case. The great danger in this type of case is in the prolapse of ileum from the umbilical opening. This complication occurred in one of the 2 cases reported by Kittle et al,[10] who found 26 other such cases making a total of 27 out of the 131 cases of patent omphalomesenteric duct they were able to collect. It is probable that the patient reported here, might have developed this complication had he not been operated upon early,

since there was a definite protrusion of ileal mucosa from the umbilicus. This was observed to be protruding more prominently at the time of operation than it had been when the patient was first admitted to the hospital. (Compare figs. 2 and 7.)

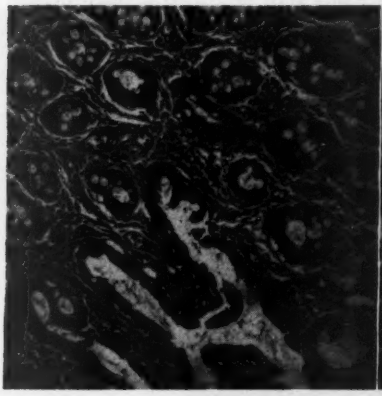


Fig. 11. Low power photomicrograph showing intestinal epithelium with deep branching gland formation, goblet cells, and a mild chronic inflammatory reaction in the stroma.

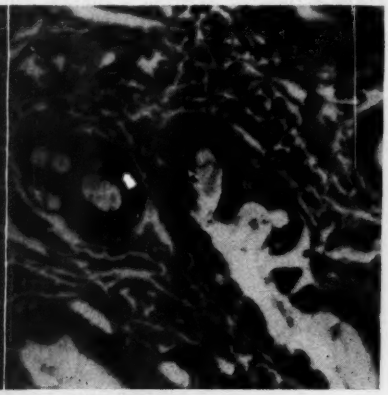


Fig. 12. High power photomicragraph of same area.

Fig. 13. Appearance of abdominal wall 12 days after operation.

Of the 131 cases of patent omphalomesenteric duct collected by Kittle et al,[10] the one reported by Crymble[4] had the origin of the duct from the appendix and was patent throughout. The case reported separately by Singleton and King[21] was also patent throughout and in contrast to Crymble's case, the end of the appendix, rather than the side or the base, formed the fistulous opening at the umbilicus. Whether these represent true Meckel's diverticula is questionable, however, Walthard[24] states that it is untenable on an embyological basis for a Meckel's diverticulum to originate from this site and attributes it to the "doubling" of a very long

appendix. The case reported here represents a true patent omphalomesenteric duct, which is a very unusual type of Meckel's diverticulum.

SUMMARY

1. Brief mention is made of historical data, embryology and incidence of Meckel's diverticulum.

2. The types of Meckel's diverticulum the heterology, and the complications are discussed.

3. Fifteen cases over a 10 year period at John Sealy Hospital are mentioned.

4. A case of patent omphalomesenteric or vitelline duct is reported.

REFERENCES

1. Christi, cited by Merritt and Rabe.[15]
2. Cobb, cited by Merritt and Rabe.[15]
3. Cole, W. H., and Elman, R.: Textbook of General Surgery, ed. 5, New York, D. Appleton-Century Co., 1948, chap. 25, p. 718.
4. Crymble, cited by Kittle.[10]
5. Curd, cited by Merritt and Rabe.[15]
6. Harbin, cited by Merritt and Rabe.[15]
7. Hildanus, Fabricius, cited by Kittle.[10]
8. Howell, L. M.: Meckel's diverticulum; consideration of anomaly, with review of sixty-one cases, Am. J. Dis. Child. *71:*365 (April) 1946.
9. Hudson and Koplik, cited by Kittle.[10]
10. Kittle, C. F.; Jenkins, H. P., and Dragstedt, L. R.: Patent omphalomesenteric duct and its relation to diverticulum of Meckel, Arch. Surg. *54:*10 (Jan.) 1947.
11. Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. S. Saunders Co., 1941, chap. 6, p. 71.
12. Lavater, cited by Kittle.[10]
13. Littre, cited by Kittle.[10]
14. Meckel, cited by Kittle.[10]
15. Merritt, W. H., and Rabe, M. A.: Meckel's diverticulum, Arch. Surg. *61:*1083 (Dec.) 1950.
16. Mery, cited by Kittle.[10]
17. Morgagni, cited by Kittle.[10]
18. Noel, cited by Merritt and Rabe.[15]
19. Ruysch, cited by Kittle.[10]
20. Schaetz, cited by Kittle.[10]
21. Singleton, A. O., Jr., and King, W. B.: Persistent vitelline duct continuous with appendix, Surgery *29:*278 (Feb.) 1951.
22. Thompson, cited by Merritt and Rabe.[15]
23. von Taignon, cited by Kittle.[10]
24. Walthard, cited by Kittle.[10]

PLASMA CELL MASTITIS

GUSTAVUS A. BYNUM, M.D.
ED. B. ROWE, M.D.
Galveston

SURGEONS and pathologists have been increasingly interested in plasma cell mastitis since it was described as a definite entity 20 years ago. Though the disease is a rarity, only 53 cases having been reported in the English literature, it is nevertheless important because it is an inflammatory condition frequently and consistently mistaken for carcinoma.[5] Pathologists have grouped this entity with the nonspecific inflammatory lesions of the breast, but surgeons have been misled because of the unusual clinical resemblance to carcinoma and have performed radical mastectomy in more than half of the reported cases.

The term "plasma cell mastitis" was first used by Ewing to describe a benign inflammatory lesion occurring in the nonlactating breast. Ingier in 1909 and Hoerz in 1910 reported a similar condition called "mastitis obliterans" now known to be identical with plasma cell mastitis. The first published report using this term was that of Cheatle and Cutler in their book, "Tumors of the Breast" (published by E. Arnold and Company, 1931). They based this report on their study of cases at the Memorial Hospital for Cancer and Allied Diseases, New York. Adair in 1933 published a detailed study of 10 cases from the Breast Service of the Memorial Hospital.[1] Further reports in the American literature appeared by Rodman and Ingleby[9] (1939), Miller[5] (1939), Cromar and Dockerty[2] (1941), Payne, Strauss, and Glasser[8] (1943), Parsons, Henthorne, and Clarke[7] (1944), Gaston[3] (1947), and Halpert, Parker and Thuringer[4] (1948). Newton[6] reported 2 additional cases in 1949 from Sydney, Australia.

Adair[1] reported 10 cases from the Breast Service of the Memorial Hospital which they had observed over an eight year period. Harrington quoted 24 cases of plasma cell mastitis in 12,000 breast lesions observed over a 30 year period at the Mayo Clinic.[2] Parsons, Henthorne, and Clark[7] reported 5 cases collected from a total of 1,500 breast cases at the Vicksburg Hospital. In 906 cases of breast tumors of all types at the John Sealy Hospital over a 30 year period there is only 1 recorded case of plasma cell mastitis.

The cause of plasma cell mastitis is not known but all authors agree that it is inflammatory in origin.[1,2,7,9,10] Bacteriologic studies have produced no more than a few unidentifiable cocci from nipple

From the Department of Surgery, University of Texas Medical Branch, Galveston

secretion in known cases. Ewing believed the causal agent to be chemical rather than bacterial in nature and due to decomposing fatty material in the ducts. Rodman and Ingleby suggested that the causal agent was derived from the products of lactation as they reproduced similar lesions experimentally. Parsons, Henthorne, and Clarke suggested that the escape of material rich in lipid content into the surrounding periductal tissue in patients who have a coexisting comedomastitis was the exciting factor.

The natural history of plasma cell mastitis can be divided into two distinct clinical stages.

A. The acute phase has an acute onset, but pain, tenderness, and discomfort are so mild that the patient does not seek medical advice. The breast is swollen and there is increased localized heat. The axillary nodes are enlarged and tender. The lesion usually does not proceed to abscess formation but gradually subsides leaving a hard painless tumor.

B. The residual phase varies in duration from several weeks to several months after the acute process subsides. The presence of a painless lump brings the patient to the doctor. Examination reveals a localized mass or diffuse thickening attached to the overlying skin. Edema is often present over the tumor or in the dependent portion of the breast giving the typical orange-skin appearance. There may be a discharge from a retracted nipple. Enlarged firm axillary nodes are usually present. Acute or subacute inflammatory signs are usually lacking although the case presented in this report showed a definite area of fluctuation which drained spontaneously the day before operation. It is the residual phase which so closely simulates carcinoma. The clinician will, in the presence of multiple signs of carcinoma, arrive at a diagnosis of malignancy unless he has carefully considered the history of antecedent acute inflammatory manifestations.[1,2]

The breast involved by plasma cell mastitis shows numerous dilated ducts and minute cysts which exude a thick creamy puriform material. The fibrous and epithelial elements are indurated with radiating strands of dense fibrous connective tissue. In some areas there may be found soft seminecrotic grey areas and xanthomatous foci. The induration and resistance of the tissue simulates carcinoma; however, in the more dense portions the classical cicatrices and chalky appearance characteristic of carcinoma are not present.[1,2,10]

The affected portions of the breast exhibit an active acute and subacute inflammatory reaction with numerous polymorphonuclear leukocytes, lymphocytes and plasma cells. This reaction is diffuse

and involves both the interstitial and the glandular tissues. The exudate is very prominent around the ducts and acini where the cellular reaction may consist almost wholly of plasma cells. Foreign body giant cells may be seen in the center of these foci. There are dilated ducts distended by epithelial debris. The epithelial cells lining the ducts undergo hyperplasia which may be so prominent as to simulate carcinoma. The presence of the giant cells may make differentiation from tuberculosis puzzling.[1,7,10]

Ewing considered the main anatomic feature to be the presence of many thickened ducts which are filled with puriform material and involve a large segment or nearly the whole breast. The most important histological feature according to Ewing is the possibility that the lesion might be considered tuberculous. Differentiation from tuberculosis, important because of the presence of giant cells, rests on the facts that in plasma cell mastitis (1) ductal and periductal tissues are involved whereas tuberculosis shows a predilection for small blood vessels and lymphatics, (2) there is no caseation, (3) axillary nodes though inflamed contain no giant cells, and (4) bacteriologic study reveals no acid fast organisms.[2,4,10]

Clinical evidence does not support the idea that plasma cell mastitis is a premalignant condition.[10] This curious, unexplained inflammatory process tends to spontaneous regression and resolution. Repeated examinations will determine whether the lesion is progressing or regressing. Satisfactory healing is the usual thing, and recurrence has not been observed.

The average age of the patient has been 30 to 40 years and all previous cases had occurred in parous women until Newton's 2 cases which occurred in nulliparous women 58 and 62 years of age. The symptom of pain was noted in 80 per cent of cases associated with localized redness and at times a discharge from the nipple. The presence of a lump was the usual reason for the patient seeking medical advice. In the acute phase tenderness was minimal. Nipple discharge was present in 70 per cent of cases. Retraction of the nipple was present in 80 per cent of the cases. Adherence to the skin was present in 60 per cent of cases, and an orange-skin appearance in 40 per cent. Nipple retraction and enlarged axillary nodes were more consistently present than in carcinoma.[10]

Proper treatment depends on a correct diagnosis. It is therefore obvious that an exact diagnosis will save many patients from unnecessarily large operations. Cheatle and Cutler have advised against surgery in the acute phase as this may cause dissemination and acute toxemia.[9,10] Since the lesion is radiosensitive, some surgeons favor irradiation in the acute phase so as to convert it to the

chronic phase before operation. Others favor biopsy section for diagnosis and simple mastectomy as the treatment of choice.

Case Report

I. S., a Negro woman, aged 38, was admitted to the Surgical Service at John Sealy Hospital on Nov. 9, 1951, at which time her chief complaint was a lump in the right breast.

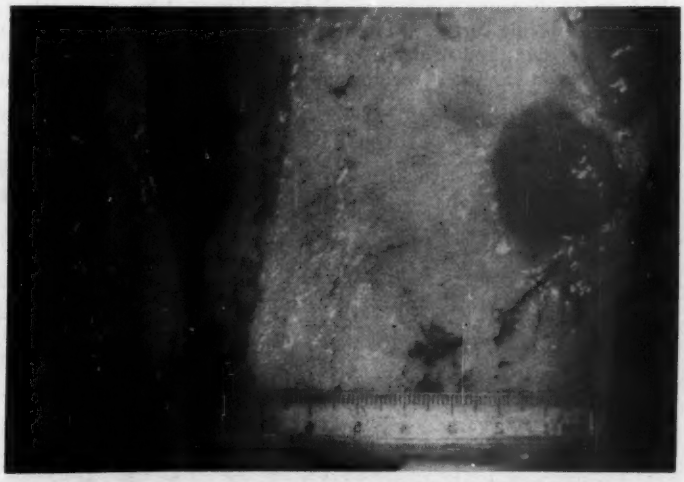


Fig. 1. Photograph of the gross specimen showing the circumscribed areas of soft, grayish-white tissue. Multiple sinuses containing similar material ramified throughout the breast.

The patient stated that she had noticed a lump deep to her right nipple for two or three years and occasionally she had noticed stinging pain near the area of the lump. There was no history of variability in size of the lump in relation to the menstrual periods. About one month previous to admission to the hospital the patient noticed that the lump was enlarging and was hot and tender, with accompanying redness of the overlying skin. There was no history of trauma to the breast. There had been no discharge from the nipple until Nov. 15, 1951, at which time a bloody discharge from the right nipple occurred. This continued and was present when the patient was admitted to the hospital.

Past history revealed that there had been four pregnancies which resulted in a therapeutic abortion for hyperemesis gravidarum in 1938, one spontaneous abortion in 1949, and two normal deliveries. There had been no difficulty with the breasts during any of these pregnancies.

Physical examination showed temperature 98 F., pulse 80 per minute, respirations 20 per minute, and blood pressure 130/88. The patient was a well developed, somewhat obese Negro woman of the stated age who did not appear acutely ill. The general physical examination showed no abnormalities with the exception of the right breast.

The breasts were somewhat large and pendulous, the right breast being slightly larger than the left. With the patient in the sitting position, a mass

could be seen in the right breast beneath the nipple and areola. On palpation, the mass was firm, very tender, about 6 by 10 cm. in size, and was attached to the nipple and the skin. The mass was directly under the nipple and areola, but extended laterally and inferiorly to the areola. No blood or fluid could be expressed from the nipple by pressure on the mass. The skin over the mass was red and warm and had an orange-skin appearance with some scaling of the epidermis around the nipple. There were several firm lymph nodes palpable in the right axilla. There was a fluctuant area lateral to the areola 3 cm. in diameter which drained 15 to 20 cc. of brown purulent material the night before surgery. The left breast was normal to physical examination. Blood count and urinalysis were within normal limits and the blood serology was negative. Chest roentgenogram and bone survey were normal.

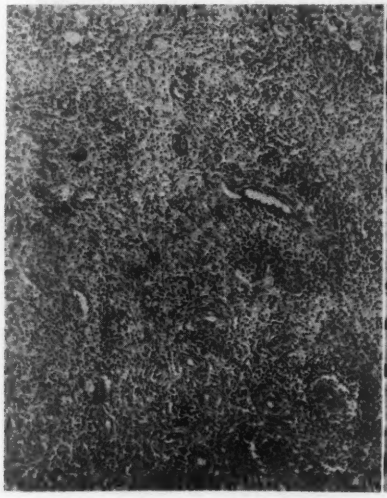


Fig. 2. Section through a representative part of the lesion. 90 X.

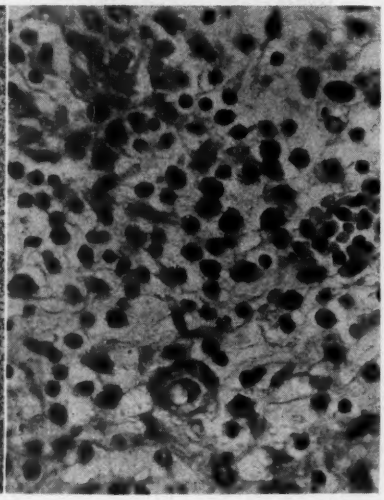


Fig. 3. Section of the lesion showing the dense infiltration of polymorphonuclear leukocytes and plasma cells. Numerous foam cells and epithelioid cells can be seen. 645 X.

The clinical impression in this case was a chronic inflammatory lesion of the right breast, most probably granulomatous in nature; however, the possibility of carcinoma was recognized and could not be ruled out by clinical means. The location and extent of the lesion made a local excision without sacrifice of the nipple impossible; therefore, it was decided to do a simple mastectomy with removal of the entire lesion. On Nov. 21, 1951, a simple mastectomy was done. A frozen section of the lesion showed it to be plasma cell mastitis. No further operative procedure was done. The postoperative course was entirely uneventful. The patient was discharged on Nov. 27, 1951. She has been seen several times since operation and remains well.

Summary

An analysis of 53 reported cases of plasma cell mastitis is given.

Plasma cell mastitis is a rare inflammatory lesion of the female breast which clinically very closely simulates carcinoma. A diagnosis can be made clinically only by careful evaluation of the history of a previous inflammatory process. The diagnosis is confirmed by biopsy and the gross and microscopic examination of the tissue. A case is presented which exhibited a sanguineous nipple discharge and abscess formation. It is suggested that simple mastectomy affords a means of diagnosis and is adequate treatment.

REFERENCES

1. Adair, F. E.: Plasma cell mastitis; lesion simulating mammary carcinoma; clinical and pathologic study with report of ten cases, Arch. Surg. *26:*735 (May) 1933.
2. Cromar, C. D. L., and Dockerty, M. B.: Plasma cell mastitis, Proc. Staff Meet., Mayo Clin. *16:*775 (Dec. 3) 1941.
3. Gaston, E. A.: Plasma cell mastitis, Surgery *21:*208 (Feb.) 1947.
4. Halpert, B.; Parker, J. M., and Thuringer, J. M.: Plasma cell mastitis, Arch. Path. *46:*313 (Oct.) 1948.
5. Miller, J. K.: Plasma cell mastitis; pathologic entity, Am. J. Surg. *43:*788 (March) 1939.
6. Newton, N. C.: Plasma cell mastitis; report of two cases, Australian & New Zealand J. Surg. *19-20:*152 (Nov.) 1949.
7. Parsons, W. H.; Henthorne, J. C., and Clark, R. L., Jr.: Plasma cell mastitis; report of five additional cases, Arch. Surg. *49:*86 (Aug.) 1944.
8. Payne, R. L.; Strauss, A. F., and Glasser, R. D.: Mastitis obliterans, Surgery *14:*719 (Nov.) 1943.
9. Rodman, J. S., and Ingleby, H.: Plasma cell mastitis, Ann. Surg. *109:*921 (June) 1930.
10. Sheehan, V., and Geoghegan, F.: Plasma cell mastitis, Irish J. M. Sc. pp. 464-469 (Oct.) 1950.

MANAGEMENT OF RADIATION INJURIES

STEPHEN R. LEWIS, M.D.
Galveston

POSTIRRADIATION injuries have seemingly not decreased appreciably in proportion to the increased knowledge of roentgen ray and radium. We can probably anticipate being confronted with more irradiation injuries with the advent of radioactive isotope work and future atomic warfare. The present day radiologist feels that multiple port and tangential use of roentgen ray with proper screening and filters should allow for treatment of even extensive malignancies using full tumorcidal dosage without injury to overlying and adjacent tissues. Yet with all this knowledge, unjustifiable radiation injuries occur from treatment of many benign and malignant lesions.

The widespread use of roentgen ray in benign lesions such as keloids, birthmarks, plantar warts, pruritis ani, keratosis, and multiple other dermatologic lesions has produced a goodly number of radiation changes which in some instances were worse than the original lesion. Sources of these injuries also have come much too often from overindulgence by physicians and dentists in their handling of the x-ray apparatus, by prolonged manipulation of fractures or foreign bodies with the fluoroscope and from multiple handling of x-ray films as in the case of the dentist who holds the film for every intra-oral roentgenogram. These burns may be either acute or chronic and should at the present time be preventable by the utilization of simple precautions.

Hyperkeratotic and malignant changes are quite common in the pioneers of the utilization of x-ray. Brown, McDowell, and Fryer[1] have presented numerous instances of these unfortunate cases.

Since the discovery of x-ray by Roentgen in 1895 and of radium by Curie in 1896, considerable knowledge has been added to the literature as to the pathogenesis and histopathologic changes. Codman's[3] hypothesis of primary injury to the nerves causing the cutaneous changes in the skin has since been discarded in favor of Wolbach's[7] study in 1909 at which time he based the response on the obliterative processes in the blood vessels of the corium and subcutaneous tissues. Further histopathologic studies by Teloh, Mason and Wheelock[6] have given extensive information on the pathologic changes occurring following extensive radiation. Sensitivity of the individual varies as to the extent of radiation necessary to cause injuries.

From the Department of Surgery, Plastic and Maxillofacial Division, University of Texas Medical Branch, Galveston.

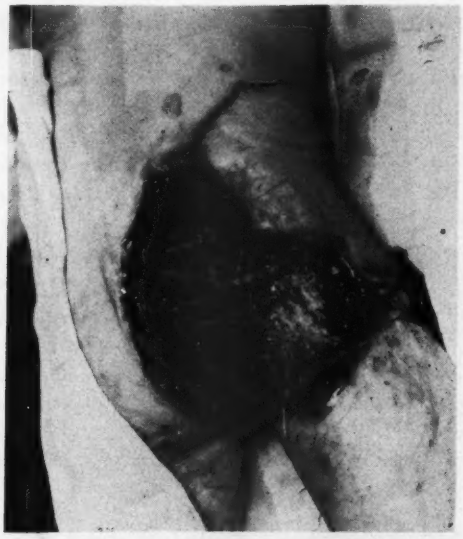
The diagnosis of radiation injuries is usually easy to make on the basis of history and the typical physical findings. However, in some instances malignant changes or bacterial and mycotic infections may distort the typical picture and confuse the diagnosis. In-

Case 1. Fig. 1. Post-burn 1948.

asmuch as the laity is not aware of injuries due to x-ray many of them present false histories of varying types of trauma to account for their lesions. Furthermore many patients receive multiple treatments such as radium, external radiation, or radon seeds by different physicians during phases of the management of a case which tends to confuse not only the patient but future surgeons who may be required to handle the case. This is a distinct plea to all physicians and radiologists to keep accurate records of therapy so that future care of an original lesion can be planned with avoidance of overdosage in subsequent management.

Acute injuries result from either a massive primary insult of prolonged exposure or from multiple exposures over a relatively short period. These are usually characterized by a reddened, edematous area with associated throbbing and severe pain. These gradually become worse for a period of approximately two weeks and then undergo a stage of healing or necrosis. In the acute phase conservative management should prevail because of the difficulty

of determining the extent of damage. Treatment at this stage should be an attempt to keep the patient comfortable by sedation plus elevation of the affected part in an attempt to limit or decrease the edema. Aloe vera used as a whole leaf may relieve some of the



Case 1. Fig. 2. December 1949 following radiation for keloids.

pain and pruritis that is associated in the acute stage. Irritants or further radiation therapy should be avoided from the onset of the burn. These acute burns follow the course of healing with some desquamation or form necrotic sloughs which can be excised and managed with replacement skin grafts.

Chronic burns may not show up for periods of many years following exposure, and often are treated by various irritants or drugs before they are recognized as radiation injuries. The management of these cases has varied considerably in the past but most surgeons now follow the methods advocated by Brown and his associates.[2] Superficial ulceration can usually be handled by simple excision and skin grafts, whereas in deep ulceration local or distant flaps (such as the blood-carrying pedicles advocated by Brown) are generally preferred. Carcinoma is prone to develop in sites of radiation damage. The progressive stages of atrophy, telangiectasis, keratosis, and carcinoma is a typical pattern if treatment is not instituted in the early stage. The vascular bed in these patients is poor and must be removed for adequate relief of pain and for complete eradication of the lesion. The vascularity has been

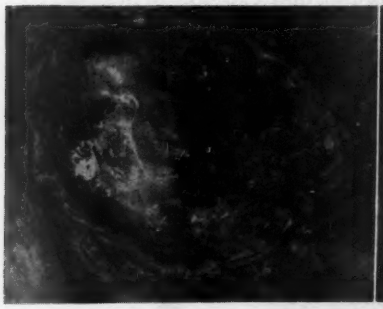
checked by the use of fluorescein by Lange.[5] By utilizing fluorescein in the method described by Lange and as advocated by Freeman[4] the extent and depth of the vascular-bed can be readily determined at the time of surgery. We have found that excision with resulting relief from pain is usually a good prognosis for adequate vascularity in the use of skin grafts or flaps. In cases where there is residual pain we have found that our excision has usually been inadequate. If one is not sure of the depth of the burn at the time of excision, it may be advantageous to wait two or three days before covering the area with the skin graft or flap. The planning program for reconstruction should be worked out as soon as possible after first seeing the patient. Evaluation as to which coverage will give the best functional repair as well as good cosmetic improvement can be determined by careful planning.

We have seen many cases of malignancies treated by radium or x-ray in other communities and then transferred to our institution for further radiation therapy. It is important in these cases to have a complete résumé of dosage and even then it is sometimes difficult to determine all of the factors necessary before further therapy is indicated. In these cases it is imperative that no further radiation be given without complete knowledge of the previous treatment

Case 1. Fig. 3. November 1950 local flap transfer and skin graft.

and even then one should hesitate before subjecting the patient to further radiation which might cause extensive damage. Where further radium therapy is indicated, we have found that elevation of a flap which is rolled away from the site of treatment and su-

tured loosely will avoid further damage to the skin and subcutaneous tissues while the radium is left in place. This rolled flap receives a minimal amount of radiation and thereby limits further

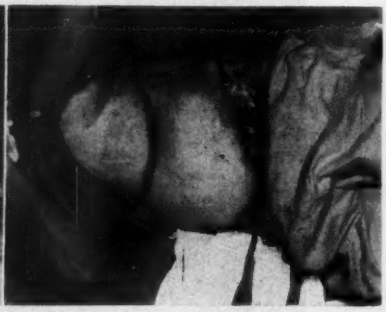


Case 2. Fig. 1. Radiation ulcer with malignant changes involving lower abdominal wall including the peritoneum.

Case 2. Fig. 2. Excision of ulcer including peritoneum showing bowel exposed.

insult. This method is also applicable in cases which have not received previous therapy for underlying malignancies where intensive dosage is desired in a relatively short period of time. This is illustrated in case 3.

Case 2. Fig. 3. Peritoneum closed with tantalum mesh, lipoma being removed at the upper aspect.



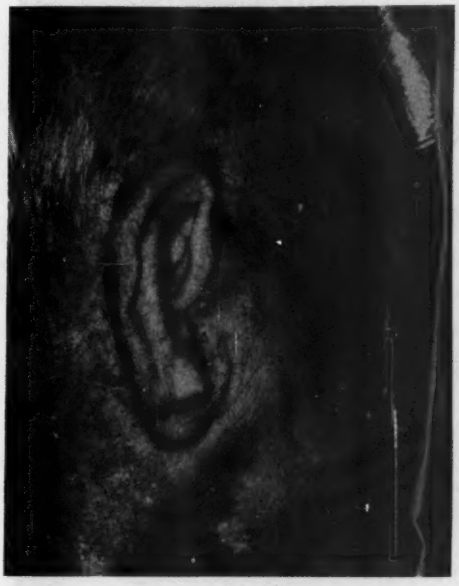
Case 2. Fig. 4. Following delay of the flap which is to be transferred over remaining site of radiation injury. Note: Sutures are not through flap edge.

Case Reports

Case 1. An 8 year old white girl was first treated for second and third degree burns of the buttocks and thighs with two skin grafting procedures in March 1948. Postoperatively she had moderate keloid-like cicatrices over both buttocks which caused some pain and pruritis. She returned in September 1949 following radiation treatment, given elsewhere, to the right buttocks with a resultant extensive ulcer. In the center of the defect, the greater trochanter, upper 3 inches of the shaft of the femur and the sciatic nerve were

exposed. Fixation of the hip joint with a moderate amount of demineralization was noted. Wide débridement followed by transfer of a full thickness skin flap and adjacent skin grafts were used to cover the defect. This course of treatment including removal of necrotic bone took a series of seven operative procedures over a period of one year's time. The later procedures in this case were the removal of further sequestrated bone not noted at the time of removing the trochanter and part of the upper femur. At the present time this girl is getting along quite well with the use of a brace.

Case 2. A 52 year old white woman was treated for cancer of the bladder with radiation therapy from 1933 to 1936 over the lower abdomen and sacral areas. At the time of her last treatment there was already beginning skin ulceration. She has had the complete metamorphisis of the chronic radiation injury. She was first seen by us 16 years after her last treatment at which time biopsy specimen showed a well differentiated squamous cell carcinoma. On February 4, the ulceration and surrounding tissues were widely excised including an area of peritoneum which was involved in the malignant process. Adherent bowel was dissected free with no involvement of the bowel noted. Peritoneal replacement was obtained by use of tantalum mesh followed by undermining of the entire upper abdominal wall and a relaxing



Case 3. Fig. 1. Preauricular mass over previously irradiated skin.

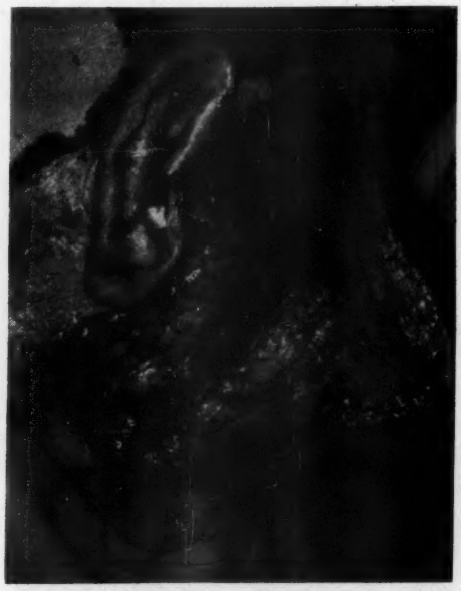
incision made in the upper abdomen to bring coverage down to the pubis. A lipoma in the right upper corner was removed at the same time. A skin graft was placed in the relaxing incision and in the left lower quadrant where we were unable to close the flap to normal tissue. Since that time we have delayed the left lateral aspect of the flap with the anticipation of swinging the

flap to cover the remaining defect. In the handling of the flap, closure is always carried out using half buried mattress sutures, thereby not coming through the superficial part of the flap and thus not further endangering its blood supply.

CASE 4. A 79 year old white man had several skin malignancies treated with external radiation therapy, a few months prior to admission. Physical examination showed a preauricular mass which was believed to be metastatic malignancy. An incision was made extending from the right temporal area along the anterior ear border to the postauricular area and below the mandible. The flap was elevated and rolled on itself and held in place by interrupted sutures. Ten 44 mm. and three 27 mm. radium needles were used with an average total of 7000 gamma rays through the entire area. Following removal of the needles on the seventh day the flap was then unrolled and resutured in its original position.

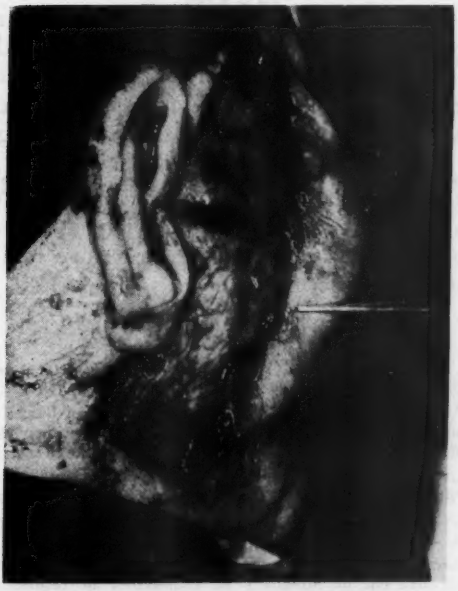
CONCLUSION

Lack of proper precautions in the use of radium or x-ray may

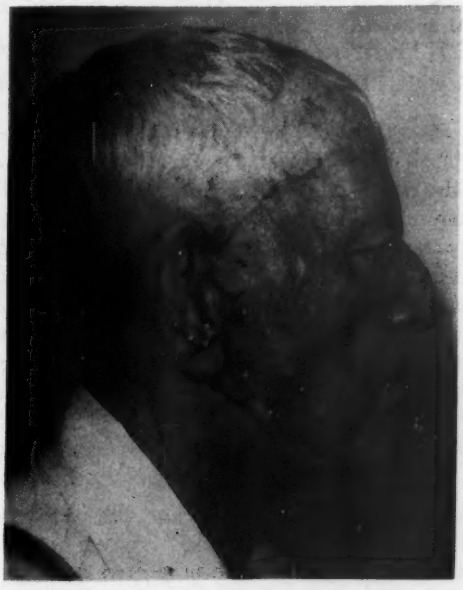


Case 3. Fig. 2. Flap elevated.

produce extensive radiation injury. Utilizing radiation therapy in a site of previous radiation is exteremely hazardous unless competent records have been carefully evaluated. A distinct plea is sent to all physicians using radium and x-ray to keep complete records so that future reference to them may prevent unnecessary injury.



Case 3. Fig. 3. Flap rolled on itself and sutured following biopsy specimen of mass.



Case 3. Fig. 4. Flap five weeks postoperative.

We believe strongly that many radiation injuries are justifiable in the management of malignancies and have frequently asked for excessive dosage in an attempt to shrink the malignancy and make surgery an easier task. This method necessitates careful planning and cooperation between the radiologist and the surgeon so that early excision and local flap coverage may be done.

One method of avoiding further injury when radiation therapy is indicated is the utilization of a rolled flap as in case 3. This method may be employed in sites where extensive radiation over a short period of time is desired.

REFERENCES

1. Brown, J. B.; McDowell, F., and Fryer, M. P.: Surgical treatment of radiation burns, Surg., Gynec. & Obst. *88:*609 (May) 1949.
2. Brown, J. B.; McDowell, F., and Fryer, M. P.: Radiation burns, including vocational and atomic exposures. Treatment and surgical prevention of chronic lesions, Ann. Surg. *130:*593 (Oct.) 1949.
3. Codman, E. A.: Study of cases of accidental x-ray burns hitherto recorded, Phila. M. J. *9:*438 (March) 1902.
4. Freeman, B. S.: Fluorescein as adjunct in treatment of radionecrotic ulcers, Surg., Gynec. & Obst. *89:*566 (Nov.) 1949.
5. Lange, K.: Vascular prerequisites of successful skin grafting; new method for immediate determination of adequacy of circulation in ulcers, skin grafts and flaps, Surgery *15:*85 (Jan.) 1944.
6. Teloh, H. A.; Mason, M. L., and Wheelock, M. C.: Histopathologic study of radiation injuries of skin, Surg., Gynec. & Obst. *90:*335 (March) 1950.
7. Wolbach, S. B.: Pathologic histology of chronic x-ray dermatitis and early x-ray carcinoma, J. Med. Research *21:*415, 1909.

THE AMERICAN SURGEON

Official Publication of
THE SOUTHEASTERN SURGICAL CONGRESS
THE SOUTHWESTERN SURGICAL CONGRESS

Published Monthly by
The AMERICAN SURGEON PUBLISHING COMPANY
701 Hurt Building, ATLANTA 3

Subscription in the United States, $8.00

Volume XVIII August, 1952 Number 8

HISTORY AND PHYSICAL EXAMINATION IN THE DIAGNOSIS OF ACUTE CONDITIONS WITHIN THE ABDOMEN

IT IS the surgeon's frequent responsibility to decide whether immediate laparotomy is indicated in the patient who has suddenly developed acute abdominal symptoms. The amount of evidence which can be brought to bear upon the decision varies with the situation. Often, in cases in which a surgical emergency actually exists, a limitation is imposed by the need to proceed without unnecessary delay. It is preferable to arrive at an exact anatomic and pathologic diagnosis before deciding upon operation, and today this can be done in a greater number of cases than formerly. Nevertheless, because of the urgent requirements attending many abdominal crises, there remains a considerable proportion in which the preoperative diagnosis can be only presumptive.

The preoperative diagnosis, as in non-emergency cases, is based upon the triad: history, physical findings, laboratory results. Thoughtful history taking and careful physical examination will supply extremely dependable information. When these two tech-

nics are applied with the skill of long practice they serve many times to indicate the diagnosis and to dictate procedure almost independent of other sources of information. In such instances laboratory findings remain important, but sometimes only to assess the patient's ability to withstand anesthesia and to tolerate surgery. For these purposes the urinalysis and complete blood count are the minimum necessities. Whether additional determinations are indicated will depend upon the surgical diagnosis and upon the presence or suspected presence of a complicating disease revealed by history and physical examination. A surgical diagnosis of intestinal obstruction will suggest immediately additional chemical and roentgenologic examinations in order to determine proper preparation and correct surgery, whereas the same tests may be completely unnecessary when the diagnosis is acute appendicitis. Again, a history of previous pulmonary disease in the patient with appendicitis may demand investigations not needed in the case with obstruction. Therefore, in acute abdominal situations of more obvious nature, one may question that laboratory procedures in excess of the minimum should ever become "routine." It is more reasonable to individualize the situation and to order only such procedures as will fulfill a definite need in the case in question.

In many acute abdominal situations the diagnosis is not obvious; history, examination and simpler laboratory tests fail to establish any definite diagnosis or to indicate whether or not immediate surgery is needed. In these "problem" cases many additional laboratory procedures may be necessary. In some obscure situations one may have to approach the problem from nearly every avenue available to gain more information. In teaching institutions with highly developed laboratory departments it is usual for exhaustive studies to be carried out in cases presenting diagnostic problems. This is justifiable for a number of reasons so long as the limits imposed by costs, by time consumed, and by the patient's welfare are observed. These are limiting considerations which must always be weighed against the likelihood of obtaining helpful information. In emergency conditions, where time becomes particularly precious, it is especially important to select laboratory procedures carefully. Fortunately, in many of the gravest emergencies the abnormal physical signs are present in such degree that they are obvious. For example, in the patient *in extremis* from asphyxia the cyanosis is so apparent that there is no reason for laboratory proof that air is needed. Similarly, in many acute abdominal cases the tenderness and rigidity are so striking that one can actually say, "This is acute appendicitis;" "This is acute cholecystitis," or "This patient has peritonitis."

The surgeon in training must not permit a justified enthusiasm for laboratory proofs to lead him to underrate the time-tested dependability of the history and physical examination. On the contrary he should develop great confidence in the patient's story and in his own palpating fingers. They will supply information just as reliable as the laboratory in many of the emergencies encountered.

ROBERT M. MOORE, M.D.
Professor of Surgery.

The University of Texas Medical Branch
Galveston, Texas

THE GALLBLADDER—A PRESSURE REGULATING MECHANISM

"Undoubtedly, the gall bladder functions as a pressure regulatory mechanism for the biliary passages. In this regard, it functions as an elastic bag connected to a system of tubes, its elasticity being a property of its elastic and muscular tunics. Its effectiveness is increased by its ability to concentrate the hepatic bile rapidly and markedly. It is a pressure regulatory and storage reservoir of small volume but of large capacity. It is able to provide for the varying rate of bile formation and for the exigency that the bile may not be able to escape into the intestine because of the resistance of the choledochoduodenal mechanism. Thus, the secretory activity of the liver may continue unhampered by the inhibitory effects of high intraduct pressures for a period of hours." (Ivy, A. C., in *Physiology Reviews,* vol. 14, p. 85, 1934.)

These are facts which are little considered by the surgeon. It is regularly observed that following cholecystectomy the biliary ducts frequently dilate. It is considered that if the biliary ducts do not dilate following cholecystectomy, then there is a relaxation of the choledochoduodenal mechanism, which prevents the building up of high pressure in the biliary ducts. Conversely, in those instances where there is irritation of the choledochoduodenal mechanism or the sphincter of Oddi and muscular wall of the duodenum, there will be a finite increase of back pressure in the biliary duct system. The magnitude of this pressure can approach the capillary pressure in the arterial capillaries, which is roughly 220 mm. of water pressure.

It is considered that bile will flow from the common duct into the gallbladder when it has attained a pressure of from 50 to 70 mm. of water. A spastic sphincter of Oddi can withstand a pressure of 300 mm. of water and the liver cells can secrete bile against a pressure of approximately 220 mm. of water pressure. It is interesting therefore to speculate on what occurs when the gall-

bladder has been removed and there is irritation in the so-called postoperative biliary dyskinesia, functional spasm of the sphincter of Oddi. The liver cells will secrete bile to maintain a pressure of 220 mm. of water which is higher than the normal pressure in the bile ducts when a functioning gallbladder is present. The result is that compensatory dilatation and hypertrophy occurs in the biliary duct system. Should the pancreatitic and biliary ducts have a common ampulla as is considered to be the case in from 15 to 20 per cent of individuals, there will also be back pressure on the pancreatic duct system. It is interesting to observe that bile obtained from the gallbladder at operation is found to contain pancreatic ferments in about 15 per cent of cases. (Nash, J.: Surgical Physiology, Springfield, C. C. Thomas, 1942, p. 186.) Therefore it is obvious that pancreatic ferments are found in the gallbladder in roughly the same proportion as a common ampulla is found between the common duct and the pancreatic duct. This observation might well mean that the normal functioning gallbladder also acts as a decompressing mechanism for the pancreatic duct system and that, following cholecystectomy, hypertrophy and compensatory dilatation due to increased pressure likewise occurs in the pancreatic ducts with the result that the accessory duct of Santorini might become of increased importance. It is conceivable that with a spastic sphincter of Oddi following cholecystectomy, bile might escape into the duodenum retrograde by the way of the duct of Wirsung hence through the duct of Santorini to the duodenum. It is an observed fact that a relatively large number of patients who have recurring acute pancreatitis have had a previous cholecystectomy. It is not inconceivable that these patients might not have had pancreatic disease prior to the removal of the gallbladder, but that this condition developed subsequently because of the imposed abnormal pressure relationships in the biliary and pancreatic duct systems.

These facts and assumptions are interesting and provocative and might well indicate that more attention should be given to the preservation of the decompressing mechanism of the biliary tract and pancreatic duct system. A study has been initiated to determine the results of internal drainage of even a diseased gallbladder by means of the Roux-Y procedure and the possible usefulness of this approach in certain diseases of the biliary tract.

EDGAR J. POTH, M.D.
Professor of Surgery.
The University of Texas Medical Branch
Galveston, Texas